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SHOCK AND ANÆSTHESIA IN RELATION THERETO.[1]

By A. DISTIN MORGAN,
Sydney.

THE subject for discussion tonight is shock and anæsthesia in relation to it. No attempt will be made to discuss the causation, symptoms, mechanism and pathology of shock; but the treatment of shocked patients will be presented, inasmuch as it is closely related to preparation for operation and to anæsthesia.

Most patients suffering from shock, whether the cause is hæmorrhage, burns, crushing or other trauma, will require surgical measures to remove the shock-producing cause. So the treatment of shock is frequently a matter of getting the patient into a fit state for operation. Only by an understanding of the methods of treatment and the response to them can be found the answers to the questions of when to operate and of which anæsthetic agent will be the safest for the patient.

Treatment of shock is no longer the responsibility of a single person, but is jointly undertaken by the surgeon, the anæsthetist and often quite a team of assistants. For the best results it is important that this team work and team spirit should be developed to a high degree. The surgeon must know the capabilities of his anæsthetist, and the anæsthetist must know the methods and speed of the surgeon. Complete confidence and understanding between members of such a team make it capable of a standard of work and success otherwise impossible.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 25, 1942.

TREATMENT.

Treatment will be discussed under the headings (i) "general" and (ii) "intravenous".

General Treatment.

General therapeutic measures are all well understood, universally accepted, and should be applied without delay in all cases of shock. They are as follows.

1. The causative factors producing shock should be removed or controlled by such measures as immobilization of fractures and control of hæmorrhage, so far as these are possible without an anæsthetic.

2. Rest can be described as the complete absence of movement, mental tranquillity, peace of mind. The importance of rest cannot be too strongly stressed. Movement of a patient is one of the most violent accelerators of shock in those who are the subject of hæmorrhage, trauma or prolonged anæsthesia. Who has not noted the rapid deterioration of a patient's condition following the movement entailed in transportation from the operating theatre to bed? Such is not the case if the patient is left in the theatre to recover. Another example is seen when a patient undergoing an abdomino-perineal resection of the sigmoid colon is turned on the side for the concluding stage of this operation.

3. The administration of adequate morphine for the relief of pain is an essential measure of treatment. The need for the adjustment of dosage must be borne in mind when there has been great blood loss, or after a prolonged period of anæsthesia or when the patient is very old or very young. Morphine dosage should never be empirical in cases of shock. Noteworthy, commenting on morphine dosage at a recent discussion at the Royal Society of Medicine, whilst fully admitting the value of morphine, said that during the raids in England many frail persons actually died of morphine poisoning.

4. Promotion of warmth, or rather the minimization of heat loss, can be assisted by the use of warm blankets, radiant heat and hot air; but by far the most satisfactory method is a special room in which both heat and humidity can be controlled. Much of the heat loss from the body takes place through the lungs, and the experiments of Professor Ralph Knight show that "body temperature can be pretty well controlled by controlling the temperature of the inhaled gas". In such a room the optimum temperature is from 70° to 80° F., with a humidity of 65%.

5. Oxygen is the primary requirement for the life of the organism, no one cell of which can survive without it. Whatever differences of opinion may exist about many aspects of shock, there is universal agreement that its end result is diminished tissue respiration—anoxia. Oxygen in adequate dosage, then, is always necessary in the treatment and prophylaxis of shock, and should be administered from the moment the patient is admitted to hospital. The employment of oxygen in the prevention and treatment of shock has received too little attention. In the past, the presence of cyanosis has been the standard accepted as the requirement for the administration of oxygen. This is as fallacious as acceptance of the blood pressure as an indication of the presence or absence of shock. Oxygen want may be extreme in the absence of cyanosis, and shock may exist to a pronounced degree in the absence of a low blood pressure. Not only is oxygen necessary in all cases of shock, but the value of high concentrations is proved. The inhalation of a 100% concentration of oxygen adds about two volumes *per centum* of oxygen in physical solution to the blood, and raises the total amount in the blood by 10% to 15%.

Clinical experience and estimations of the oxygen content of the blood have proved conclusively that of the ordinary methods available for oxygen administration, the "B.L.B." mask and the nasal catheter are the most efficient and economical. The "B.L.B." mask is available in two patterns for the continuous administration of humidified oxygen. For conscious patients, the nasal type is designed for nose breathing, and a space is provided to give access to the mouth for the administration of fluids and food. For unconscious patients, or when nose breathing is impossible, the oro-nasal type, which covers both the nose and the mouth, is the better one to use. Humidification is provided by the partial rebreathing bag. An oxygen flow varying between four and eight litres per minute will give 100% oxygenation, according to the age, sex and weight of the patient. The same rate of flow through a nasal catheter will give 50% oxygen, provided the catheter is correctly placed. The tip should be in the oro-pharynx, just beyond the edge of the palate, and above the base of the tongue and epiglottis. A rough estimate of the correct length of the catheter is the distance from the tip of the nose to the lobe of the ear. Lubrication is necessary, and one of the anæsthetic applications may be used for this purpose. If the catheter is brought across the cheek, there is a tendency for it to touch the upper and more sensitive parts of the nose, thus causing irritation and discomfort. If, on the other hand, the catheter is brought over the tip of the nose and up to the forehead, it tends to lie still on the floor of the nose. In this position, it causes no discomfort, and even children will tolerate it for days without complaint. Prolonged administration of dry oxygen through a catheter will cause drying and damage to the epithelium of the upper air passages. A simple and inexpensive humidifier and flowmeter can be made after the pattern designed by Professor Ralph Waters. Many other methods of administering oxygen have been used, but compared with those described, they are either inefficient or too costly, or both.

In blast injury, in which the alveoli of the lung are filled with blood and exudate, there is lessening of the exchange of oxygen from the alveolar air to the blood. In these cases, the subcutaneous and intravenous routes for the administration of oxygen may have a useful place.

6. The oral administration of warm, sweetened drinks provides an excellent means of giving fluids to conscious patients who are not vomiting. Continuous rectal drip administration of water can be quite efficient if details of technique are carefully observed. A fine, soft, well-lubricated catheter, inserted for a distance of six inches and kept in place with strapping, will be tolerated for as long as three days. When the drip rate is 20 per minute, two to two and a half litres will be absorbed in twenty-four hours; this amount will go a long way towards maintaining fluid balance, and will serve as a prophylactic measure for patients not able to take sufficient fluid by mouth. It is well to recall that fluids administered by the rectum enter the blood stream via the portal circulation, where they collect plasma proteins. Consequently, they do not lessen the concentration of plasma proteins. It is my experience that glucose and salt solutions are not absorbed so readily from the rectum, and that if they are used they will lessen both the volume absorbed and the time for which the catheter will be tolerated.

Intravenous Treatment.

In all cases of severe shock the intravenous route is necessary for the administration of fluid, first, because quantities are required which could not be given in the time by any other route, and secondly, because fluid such as blood, serum or plasma is of the utmost importance.

The vast amount of new knowledge about shock which has been brought to light in recent years is reflected in our changed, and still changing, ideas about intravenous therapy. Whereas only a few years ago normal saline solution and blood were used, there are now available many fluids which can be employed. To mention only some of the additions, there are now several strengths of hypertonic saline solution, glucose solutions varying from 5% to 25%, serum, plasma, and the dried products from which we can make concentrated solutions of plasma proteins. Each of these fluids produces its own effect.

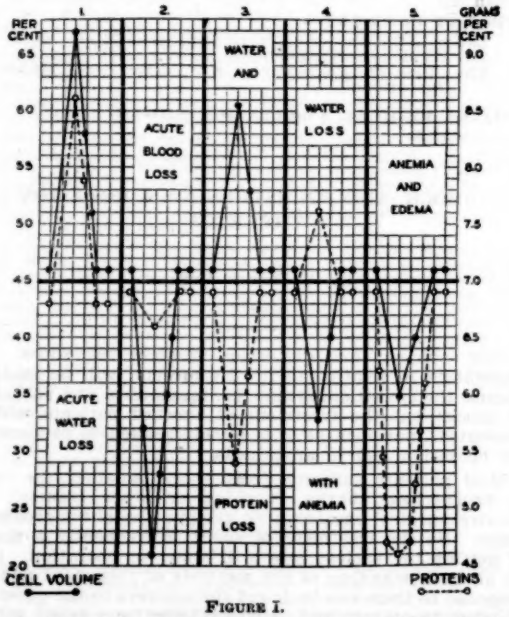

FIGURE I.

C. R. Drew's hæmatocrit-protein patterns. (Reproduced from *Anæsthesiology*, Volume III, Number 2, 1942, page 176.)

The employment of some special fluid for intravenous therapy in shock requires an understanding of the exact condition of the patient. In the diagnosis of the type and degree of shock present, ordinary clinical methods are of little use. Some information can be obtained from the history, the general appearance and an ordinary clinical examination. The nail bed reflex can give information about the degree of stasis present in the capilliary bed. A falling blood pressure and a rising pulse rate indicate rapid deterioration of the patient's condition. Conversely, a rising blood pressure and a falling pulse rate indicate improvement. The compensatory constriction

of the vessels and of the spleen keeps the blood pressure within normal limits, long after permeability of the capillary endothelium and hæmoconcentration prove the existence of shock. Therefore, little reliance can be placed on a normal blood pressure reading when the presence of shock is suspected.

The information required for both diagnosis and treatment is quantitative; it is given by (i) the percentage of cells in the venous blood (hæmoconcentration) and (ii) the protein content of the whole blood and of the plasma.

Hæmoconcentration is measured by the hæmatocrit, and a sufficiently accurate reading can be obtained in ten minutes. A blood count and a hæmoglobin estimation also give an indication of the hæmoconcentration, but are less reliable. The plasma protein content is calculated by a formula from the specific gravity. So far, we have not been able to make this estimation quickly enough for it to be of value in diagnosis, but it is hoped in the near future to obtain apparatus which will provide readings in a few minutes.

Dr. Charles R. Drew described these tests, and the results from a large series of cases of shock, in a paper presented before the American Society of Anesthetists. He found that the hæmoconcentration and protein values are remarkably constant in cases of shock from the same cause. He demonstrated well-defined patterns, which, when considered along with the history and clinical findings, clearly indicate the line of treatment.

Treatment of Shock Associated with Various Lesions.

From these patterns, and from clinical experience in the treatment of patients suffering from shock, it is apparent that a definite line of treatment can be laid down in certain cases. The treatment of such cases will now be considered.

Acute Blood Loss.

In acute blood loss we have a definite clinical picture of shock, a low cell concentration and a plasma protein content within normal limits. These facts, taken in conjunction with the history, should leave no doubt as to the treatment. A blood transfusion is required, and the amount given should be sufficient to raise the hæmoglobin value to 10 grammes per 100 cubic centimetres of blood (about 60%). Each 500 cubic centimetres of blood can be expected to raise the hæmoglobin value by 10%; that is, if the hæmoglobin value is 20%, 2,000 cubic centimetres will be required to raise the level to 60%. The rate of administration varies. For patients requiring urgent operation, such as those suffering from a ruptured ectopic pregnancy, the first 500 cubic centimetres should be run in as rapidly as possible, and then the rate should be regulated by the improvement in the patient's condition. For patients not requiring urgent operation, a rate of 20 drops per minute for the total amount is adequate. In these cases a rapid exhibition of blood may start further bleeding. The slow drip administration has other advantages, particularly when large quantities of blood are to be given. It avoids overloading of the right side of the heart, and it produces a higher rise in the hæmoglobin value than does the same quantity of blood given rapidly.

Burns, Peritonitis et cetera.

In burns, peritonitis from ruptured ulcers, or infection resulting in the pouring into the peritoneal cavity of large quantities of fluid rich in protein, increased cell volume and great protein loss are present. The area of the peritoneum is much the same as the skin area, and protein loss from the circulation can be very considerable, either from the skin in the case of burns or from the peritoneum in this type of peritonitis. This, however, is not the only source of fluid loss and protein loss from the circulation. It has been shown experimentally in animals that burnt skin, or peritoneal fluid when introduced into the peritoneal cavity of a healthy animal, will cause shock, with diminished circulating blood volume, cell concentration and diminished plasma protein content, resulting in death.

Treatment is by plasma or serum given in large amounts, as much as four litres being necessary in as many hours. Saline solution should not be used intravenously in these cases, as it further increases fluid loss from the seriously damaged capillary bed to the tissues and decreases the protein content in the plasma to the level at which œdema follows. Blood raises the plasma protein concentration, but also increases the cell concentration and the viscosity of the blood. If serum or plasma is not available, blood is next best.

Acute Water Loss.

Acute water loss occurs in dehydration from lessened intake or excessive loss of fluid (diarrhœa, vomiting, sweating) or in shock of traumatic or post-operative origin. There is an increase in cell volume, together with an increase in the plasma protein content. The condition may be sufficiently severe to cause acute shock, but is more frequently in existence as a pre-operative condition, and is not recognized till the added strain of surgical measures and anæsthesia have increased its severity. Treatment is by the intravenous administration of saline solution, with or without the addition of glucose. The use of blood, plasma or serum is contra-indicated, as each of them would tend to make the condition worse by increasing the cell concentration and the plasma protein content. The amount will vary according to the severity of the condition; it can really be judged only by the response to treatment. In mild cases one litre given rapidly, followed by a second litre dripping in over a period of four hours, will often prove sufficient. In more severe cases further treatment will be necessary.

Anæmia and Dehydration.

Anæmia and dehydration are seen together in such conditions as pyloric stenosis with vomiting and malignant disease of the colon with diarrhœa. They too produce a low-grade type of shock, which if unrecognized and untreated in the pre-operative period will, after the prolonged surgical procedures needed in these cases, become manifest as shock of such severity as may well prove fatal. From the point of view of treatment, the low cell concentration produced by anæmia is the important factor, and blood must be given before operation in quantities sufficient to raise the hæmoglobin value and number of cells to normal limits.

Experience has shown in cases of this nature that adequate preparation for operation is simple and effective. The same or even larger quantities of blood given after operation, when the patient is in a state of acute shock, and when pronounced capillary stasis is present, is often attended by unsatisfactory results and even by death. The recognition of these cases of mild shock is of the utmost importance, and in this connexion it can safely be said that a little prevention is worth a lot of cure.

Other Lesions.

There are other conditions about which our knowledge is still far from complete. In this group will be considered such injuries as crushing injuries, multiple wounds or fractures of the limbs, or both in conjunction, and head and chest injuries. In such cases shock of the utmost severity may be present. Clinical observation and the tests enumerated earlier have failed to produce regularly information which would suggest a definite line of treatment in any particular group. Any or all of the causative factors producing shock may be operating in this type of injury.

In this connexion the tests already referred to will prove of the utmost value in diagnosis. The present state of our knowledge would suggest that four conditions are present: (i) decreased circulating volume of blood, (ii) constriction of arterioles and venules, (iii) capillary stasis, (iv) loss of fluid into the tissues (hæmoconcentration). Treatment must be directed towards the correction of each of these.

There is no substance known which will specifically release the spasm of small vessels. However, it is known that in these cases there is a loss in the plasma content of sodium, in spite of the increased cell volume. It would, therefore, seem reasonable to commence treatment by the intravenous injection of normal saline solution. This will increase the circulating volume and may be expected to release the spasm of the vessels.

Suprarenal cortical extract, marketed as "Eschatin", when used in sufficient dosage, has a pronounced effect in restoring capillary tone and governing the distribution of electrolytes. By the means described we should have corrected the first three pathological conditions present, and it now remains to maintain in the circulation the fluids which have been added and restored. This will be best achieved by the introduction into the blood stream of a substance with an osmotic tension sufficient to prevent further loss to the tissues—namely, blood, plasma or serum. In most cases plasma or serum will prove superior to blood.

As a guide to treatment in the cases considered under this heading the following are suggested as methods which could be employed in the absence of such quantitative tests as would give exact information.

1. If serum is available, this should be used at once. The rate of administration of the first 1,000 cubic centimetres should be rapid; 500 cubic centimetres should be given as fast as the serum will flow; then it should be given at the rate of 100 drips to the minute.

2. If serum or plasma is not available, it would be unwise to give blood in the first instance, when severe hæmoconcentration is thought to be present, as this would further accentuate it. The following régime is suggested: (*a*) one litre of normal saline solution, given as rapidly as it will flow; (*b*) 250 cubic centimetres of 5% saline solution containing 10 or 20 cubic centimetres of "Eschatin"; (*c*) 1,000 cubic centimetres of blood. The normal saline solution, given rapidly, will increase the volume of circulating blood; the concentrated saline solution and "Eschatin" will replace the lost sodium and restore the tone to the capillary bed. Finally, the blood can be expected to raise the osmotic tension of the circulating fluid and prevent further loss to the tissues. No time will be lost if this suggestion is followed, for the typing and collection of blood can proceed while the saline solution is being given.

General Discussion of Treatment.

The first volume of *War Medicine* summarizes the treatment of shock in these terms:

1. Begin treatment before the condition has progressed to the point where it is irreversible.

2. Correct blood concentration and prevent further fluid loss.

3. Maintain adequate oxygenation of the tissues until the patient's own improved circulation can take care of this.

The time factor in the treatment of shock is recognized as being of the greatest importance. The least delay may well prove fatal in severe cases, and in hospitals likely to receive patients suffering from shock a complete shock team and all necessary equipment should always be ready for immediate use.

In any hospital handling numbers of casualties, either in civilian or in wartime practice, a shock ward would be a useful addition to the ordinary equipment. Some of the suggested requirements for such a ward are as follows: (i) a position adjoining the operating theatres; (ii) adjustable temperature and humidity; (iii) oxygen, available at the head of each bed from a low pressure pipe line; (iv) personnel for carrying out diagnostic and other blood tests, such as (*a*) blood grouping, (*b*) hæmatocrit readings, (*c*) tests for specific gravity of blood and plasma, (*d*) blood counts, and (*e*) hæmoglobinometry; (v) personnel for the administration of intravenous therapy; (vi) adjoining rooms for (*a*) the tests listed above, (*b*) the taking of blood from donors *et cetera*, (*c*) storage of fluids and equipment for intravenous therapy.

Renal failure has frequently followed crushing leg injuries. It would appear that the cause is anoxia of the various kidney cells. This causes damage which leads to transudation of plasma and blood cells, bringing about cessation of cell function. If this is so, then the measures listed earlier will prove the best prophylactic. The causative factor producing the shock will be removed by operation, which should be carried out at the earliest possible moment permitted by the patient's condition. This moment should be within three hours, if the patient is capable of recovery. As a further prophylactic against renal failure or as a measure of treatment once the condition is evident, sodium sulphate should be used; it is administered intravenously as a 4·28% solution, at a rate of 100 drops per minute. After less than a litre has been given the patient is generally secreting and passing urine.

Some practical points may be mentioned in connexion with intravenous therapy for shock patients. In severe cases veins are collapsed, and difficulty is experienced in introducing a needle, even if a vein-seeker is used. The time taken to cut down on a vein and insert a large blunt needle or cannula is well repaid by the certainty of results and by the absence of leakage. When the administration of the fluid is commenced the constriction of the vein may be so great as to prevent flow. This is overcome by placing the container as high as the tubing will allow, thus gaining all possible assistance from gravity, and by "milking" the fluid from the tube into the vein for a minute or two. Once the flow has commenced, it will increase in rate as the vein relaxes. This increase can be expected to continue while the patient's condition continues to improve, and to some degree is an indication of the response to treatment.

When to Operate.

It will be seen from the foregoing remarks that the response to treatment is the only guide to the important question of when to operate; though improvement is apparent, it can be at best only partial or temporary whilst the shock-producing factor is still operating. Perhaps the answer to the question can best be indicated by the discussion of a few specific cases.

In shock from hæmorrhage, after receiving 500 cubic centimetres of blood as rapidly as it will run, the patient should be ready for operation in twenty minutes. The flow of blood is continued during operation at 60 drops per minute and after operation at 30 drops per minute. The rate of flow can at any time be increased should deterioration of the patient's condition require it.

When multiple compound leg injuries are the causative factor a longer period will probably be required before operation is possible; but the time to operate is the first moment at which the patient's condition will permit anæsthesia for long enough to allow the surgeon to accomplish the minimum requirements. Team work plays a large part in these circumstances. A state of complete understanding will make it possible to commence an operation earlier than would be possible were such understanding not present. The only way that such team work can be developed is in the hard school of experience.

When obstruction low in the alimentary canal is the shock-producing factor, I have known operation delayed for as long as three days with advantage to the patient. The condition of a patient, who was admitted to hospital in a state of extreme shock, improved with shock therapy and duodenal siphonage, which were continued till the operation.

The important point for consideration is whether the causative factor is still active or whether it can be controlled. In cases in which it cannot be removed or controlled without operation the necessary operation must be undertaken as early as the condition of the patient will permit. But if the cause can be removed or its effect controlled, then operation should be delayed till the maximum improvement in the patient's condition has been achieved by treatment.

THE CHOICE OF ANÆSTHETIC AGENT.

The end result of shock, whatever the cause may have been, is diminished tissue respiration—anoxia. The first requirement for an anæsthetic agent to be given to a patient in even a slight degree of shock is oxygen. Thus, generally speaking, the anæsthetic agent which permits the use of the greatest amount of oxygen will prove the safest. During the actual administration the anæsthetist's main care will be to secure and maintain a clear airway. This will be best achieved by the careful removal of débris from the upper air passages and by the passing of a Magill tube of adequate bore under direct vision.

Cyclopropane permits of the use of the highest percentage of oxygen and is the anæsthetic agent of choice in many cases. But the drug is costly and it requires a trained anæsthetist and an expensive machine to make possible the use of the closed circuit technique. It has, therefore, a limited usefulness.

Nitrous oxide and oxygen will produce a depth of anæsthesia sufficient for minor procedures. For longer or major procedures it does not allow sufficient oxygen for safety and does not produce sufficient relaxation. The addition of ether in minimal quantities to nitrous oxide and oxygen will provide a depth of anæsthesia sufficient for any surgical procedure and will permit the use of high percentages of oxygen. The speed and smoothness of induction of anæsthesia by the nitrous oxide, oxygen and ether sequence are probably its only advantages over plain ether and oxygen. In the hands of the majority of anæsthetists the two last mentioned will probably be the most used.

Every anæsthetist will have his own preference, and it is well to remember that well-given ether and oxygen are safer than badly given nitrous oxide or cyclopropane. I would therefore suggest that, providing sufficient oxygen can be given and the airway kept open, a safe rule is to employ the anæsthetic agent you know best for use in shock.

In my own experience I have had excellent results from the use of "Pentothal Sodium" with the addition of oxygen. In some cases a Magill tube has been employed to maintain a good airway. When necessary this is introduced under local anæsthesia before the intravenous injection is begun. The main advantages of this technique are the speed of induction without excitement, the ease of control and the absence of post-operative vomiting. When the depth of anæsthesia required is greater than that afforded by this drug (for example, in upper abdominal surgery), local infiltration and field block may be added. "Pentothal Sodium" may be conveniently given into the tube delivering saline solution or other fluid to the patient. Atropine, in doses of $^1/_{100}$ or $^1/_{150}$ grain, should always precede the administration of "Pentothal Sodium". When this anæsthetic agent is used in shock it must be kept clearly in mind that less will be needed than in ordinary cases and that detoxication and excretion will be retarded. Experience with a cooperative surgeon has shown that surprisingly small doses are adequate and that the patient's condition suffers very little.

Local infiltration and field block in expert hands have a wide range of usefulness, either alone or combined with general anæsthesia. The high incidence of post-operative chest complications following the use of this form of analgesia should be borne in mind, and special measures should be adopted to guard against them. Oxygen should be administered during operation, and "Carbogen" together with early movement should be used after operation.

"Avertin", chloroform and spinal analgesia are contra-indicated for patients suffering from shock.

ANÆSTHETIC PROCEDURES FOR SOME SPECIAL TYPES OF TRAUMA.

War Injuries of the Chest.

The anæsthetic technique in war injuries of the chest is the same as that frequently used for lung surgery in civil practice. It is called controlled respiration, and it means that the anæsthetist, having abolished the active respiratory efforts of the patient, maintains an efficient tidal exchange by rhythmic squeezing of the breathing bag. Though I have used controlled respiration, my experience with it is limited, and as I have had no experience of war injuries of the chest, the substance of my remarks on this subject is taken from the excellent article by Nosworthy, whose experience is considerable.

Active respiration will cease (i) if the patient's CO_2 is lowered sufficiently by hyperventilation, (ii) if the patient's respiratory centre is depressed sufficiently by sedative and anæsthetic drugs, and (iii) by a combination of (i) and (ii).

Controlled respiration needs a closed circuit, with efficient carbon dioxide absorption, and the use of an intratracheal tube fitted with an inflatable cuff to prevent leakage of gases and inhalation of débris. This is a highly technical form of anæsthesia, requiring the services of a skilled anæsthetist.

Physical examination and X-ray pictures are essential in war casualties. One man had a collapse of the right lower lobe and a right pneumothorax without any external injury (X-ray). Without physical signs and X-ray evidence in the early stages the diagnosis of "blast" is difficult to make. Some patients, however, require urgent operation before the possibility of blast can be excluded.

Although a simple hæmothorax is treated best by aspiration, with or without air replacement, patients with a compound hæmothorax frequently require operation because the blood has clotted or in order to remove a foreign body, to secure a bleeding intercostal vessel, to close a sucking wound or to remove shattered rib fragments. By early operation normal physiological function may be restored and the patient saved from chronic infection in the pleural cavity or lung.

By means of this type of anæsthesia the airway can be kept open, adequate oxygen can be administered, early surgical treatment may be undertaken, and when the work is completed and the cause of the shock has been removed the lung can be inflated. These are important points not only in the saving of life in these desperate cases, but also in preventing the incidence of secondary infection, with its long convalescence and high mortality rate.

Acute Intestinal Obstruction.

In connexion with acute intestinal obstruction there are two problems for the anæsthetist: vomiting and distension.

It is the anæsthetist's care to see that no débris is inhaled. If the stomach is distended and large amounts are being vomited, it is necessary to empty the stomach before the induction of anæsthesia. This measure may considerably add to shock. If the amounts of vomitus are small, it is wiser to omit the wash-out and rely for induction on a rapidly acting agent, such as "Pentothal Sodium", then deepen the anæsthetic level and pass an endotracheal tube fitted with an inflatable cuff. Once this is in place, the administration of the anæsthetic agents, such as nitrous oxide, oxygen and ether, may proceed with safety. Ample relaxation may be procured, adequate oxygen may be given, and there is no danger of the inhalation of vomitus.

If distension is the predominant feature, it may be well to consider employing spinal analgesia, this being the one exception that proves the rule about not using spinal analgesia in shock. Spinal analgesia can be expected to produce both excellent relaxation of the abdominal wall and stimulation of the tone of the unstriped muscle of the gut. If this technique is adopted, the cough and swallowing reflex will be retained. The administration of oxygen and intravenous therapy should be continued during and after operation.

CONCLUSION.

I offer my thanks for the honour of being asked to open the discussion tonight. The whole subject has great interest for the medical profession at any time, but in the days through which we are passing now it has added interest and importance. There are aspects of the subject about which our knowledge now seems to be complete, and in such instances I have endeavoured to be definite in my statements. There are, however, many aspects of shock in which our knowledge is still far from complete, and about which it is impossible at this stage to do more than suggest principles that may prove useful.

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ON THE CALCULATION OF THE HEAT AND MOISTURE DISSIPATED FROM THE BODY BY RESPIRATION, WITH A TABLE DESIGNED TO MAKE CALCULATION EASY AT ANY TEMPERATURE, ANY HUMIDITY, AND ANY PRESSURE OF THE AIR.

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THIS paper contains a table (Table I) designed to make it easy to calculate the amount of heat, or of moisture, lost from the body by respiration in various circumstances.

Certain fundamental measurements are required, and the first is the volume of the respiratory ventilation current per unit of time. We require also the temperature, the humidity, and the barometric pressure of the air. How these measurements are carried out is not within the function of this paper. The degree of accuracy obtainable by the calculations will necessarily depend on the degree of accuracy reached in the original measurements.

In breathing air, one breathes usually an atmosphere only partially saturated with water vapour. It is customary to state the degree of humidity as a percentage of the full amount possible—for example, as 20%, 50%, and so forth, or, more briefly, as 20 or 50. A little consideration shows that this is equivalent in fact to an atmosphere which is a mixture of two parts, one part completely saturated, the other part completely dry, in the proportions indicated.

The air inspired has to be warmed from its initial temperature to 33° C., the temperature of respired air, and at that temperature it is saturated with water vapour, being expired as saturated air at 33° C. This applies both to the completely saturated and to the completely dry percentage moieties that are inspired. The former can, of course, receive no more moisture at its initial temperature, but as soon as its temperature is raised it becomes capable of holding more. If its temperature is raised to 33° C. it can absorb from the respiratory passages whatever amount that weight of air can hold at 33° C., less the moisture it had at first. On the other hand, if it starts at a temperature higher than 33° C., it is cooled to 33° C., and the weight of vapour carried has to be reduced to the amount that can be carried by saturated air at 33° C. The completely dry moiety of the inbreathed air receives the whole of its water vapour from the respiratory passages. It has also to be warmed or cooled to 33°C. So, too, have the vapour-free content of the saturated moiety and its vapour.

Respired air, being at a certain temperature and at a certain degree of water content, always the same, has always the same weight per cubic metre at standard barometric pressure. Its weight, however, will be altered by variations in the barometric pressure.

The requirements of the body for oxygen must be met, whatever the atmospheric pressure, and so the physiological apparatus adjusts itself automatically to differences of pressure by commensurate changes in the volume of the respiratory current. Therefore, for a given oxygen need, a given weight of air will always be obtained, though the volume of air breathed may be different. In measuring the volume of air respired through different activities, it is necessary to note the barometric pressure, and the volume should be corrected as for standard pressure, 760 millimetres of mercury or 1,000 millibars, according to the standard adopted. It is obvious that a respiration current observed at sea level cannot be properly compared with one made at a height of 1,000 metres without this correction.

If this correction is always made, the recording may be made in terms of volume—so many litres per minute, or cubic metres per day, and so forth, with the calories corresponding to that volume. But if not, the recording must be made in terms of weight—so many grammes or kilograms of air, and the calories corresponding to that weight. The table (Table I) is constructed on a volume unit—that is, to one cubic metre; but it would be easy to convert it to a weight unit if desired.

The figure used for the standard atmospheric pressure is 1,000 millibars, which is almost exactly equal to 750 millimetres of mercury. Since 760 millimetres of mercury correspond to 1,013·3 millibars, anyone wishing to use the table and express his results as standardized to 760 millimetres of mercury must multiply figures given in Table I by 1·0133.

The specific heat of dry air is taken as 0·2375, and that of water vapour as 0·4. The latent heat of vaporization of water at 33° C. is taken as 580 small calories, or 0·58 of a large calorie.

The special meteorological data used are from the Smithsonian Meteorological Tables, fourth revised edition, 1918 (hereafter referred to as S.M.T.), as far as possible, extended by personal supplementary calculations at both ends of the temperature scale, where the Smithsonian tables did not reach far enough. The Smithsonian tables are calculated to a standard pressure of 760 millimetres of mercury, so they had to be corrected, as required, to 1,000 millibars.

The Construction of Table I.

We calculate on the basis of figures in Tables 98, 99, and 100 of S.M.T. that at 33° C. and at 760 millimetres of

mercury pressure, one cubic metre of air at saturation point weighs 1,131·7 grammes. By Table 74 of S.M.T. we find that 35·656 grammes of water vapour must be present, and therefore, if we subtract this, we get 1,096·04 grammes as the weight of the vapour-free fraction.

The formula is as follows:

Let δ be the weight of a cubic centimetre of air expressed in grammes.
Let B be the atmospheric pressure in millimetres.
Let e be the pressure of aqueous vapour in millimetres.
Let t be the temperature in Centigrade degrees (t = 33° C. in the present calculation).
Let Δ be the weight of a cubic metre of air in grammes (1·0 cubic metre = 10^6 cubic centimetres).

Then:

$$\delta = \frac{0{\cdot}00129305}{1 + 0{\cdot}00367t}\left(\frac{B - 0{\cdot}378e}{760}\right)$$

= 0·0011317 = weight of 1·0 cubic centimetre.

Δ = 0·0011317 × 10^6
= 1,131·7 grammes
= weight of 1·0 cubic metre, saturated, at 33° C. and 760 millimetres of mercury pressure.

If a cubic metre of saturated air at 33° C. and 760 millimetres of mercury (1,013·3 millibars) contains 1,096·04 grammes of vapour-free air, how much vapour-free air would it contain at 1,000 millibars?

The calculation is

$$\frac{1{,}000 \times 1{,}096{\cdot}04}{1{,}013{\cdot}3} = 1{,}081{\cdot}65 \text{ grammes.}$$

If a cubic metre of saturated air at 33° C. and 760 millimetres of mercury (1,013·3 millibars) contains 35·656 grammes of water vapour, how much would it contain at 1,000 millibars?

The calculation is

$$\frac{1{,}000 \times 35{\cdot}656}{1{,}013{\cdot}3} = 35{\cdot}188 \text{ grammes.}$$

If the latent heat of vaporization at 33° C. is 0·58 large calorie per gramme, how many calories are absorbed by vaporization of 35·188 grammes at the same temperature?

The calculation is

$$0{\cdot}58 \times 35{\cdot}188 = 20{\cdot}409 \text{ large calories.}$$

The weight of the vapour-free fraction of one cubic metre of respired air is always 1,081·65 grammes (or 1·08165 kilograms) at 1,000 millibars of pressure, and it is at a temperature of 33° C. But this amount of 1,081·65 grammes was at a different temperature originally, when it was inspired, and it had to be warmed or cooled, as the case might be, to 33° C. The specific heat being 0·2375, it takes 0·2375 small calories per gramme, or 0·2375 large calories per kilogram, per degree Centigrade of temperature, to warm, or in reverse, to cool.

To find the heat required to change to a different temperature, if T_{II} is the new temperature and T_I is the original temperature in degrees of the absolute scale, the calculation will be (to express it in large calories):

$$(T_{II} - T_I) \times 0{\cdot}2375 \times 1{\cdot}08165$$

If $T_{II} - T_I$ forms a term with a negative sign, it will mean that the air has to be cooled instead of being warmed. We can write $T_{II} - T_I$ as t, and in calculating for a table one makes use of the fact that the second and third terms are constants and can be simplified to make a single constant term k.

Illustrative Example.

For example, take the calculation for temperature 20° C.

Let C_I be the heat required, in large calories.
Let a be the weight of the dry fraction of respired air at 33° C. and 1,000 millibars (1·08165 kilograms).
Let θ be the specific heat of air (0·2375).
Let t be the number of degrees of temperature to be traversed (13).
$k = a \times \theta$

Then $C_I = a\theta t$
$\log C_I = \log a + \log \theta + \log t$
$= \log k + \log t$
C_I = 3·339 large calories.

The calculations in this series form column 2 of Table I.

The next step is to find what weight of water vapour would be carried by 1,081·65 grammes of dry air at a series of given temperatures if fully saturated for each given temperature.

From the formula already given, namely,

$$\delta = \frac{0{\cdot}00129305}{1 + 0{\cdot}00367t}\left(\frac{B - 0{\cdot}378e}{760}\right)$$

we calculate (by $\delta \times 10^6 = \Delta$) the weight of one cubic metre of air saturated at that temperature and at 760 millimetres of mercury pressure. Table 74 of S.M.T. gives the weight in grammes of water vapour present in one cubic metre at 760 millimetres pressure, so we obtain the weight W of the dry fraction by subtracting the weight h of the vapour from Δ.

Then, by proportion, we can find the weight of vapour (or its calorie equivalent) that would be carried at the same temperature by 1,081·65 grammes of dry air if fully saturated. This will be the same at all pressures, so no correction for pressure is needed.

Let us find now the weight of vapour that would be carried by 1,081·65 grammes of dry air at 20° C.

From the data in S.M.T., Tables 98, 99, and 100, we calculate that Δ = 1,194·1 grammes at 20° C. Table 74 gives 17·3 grammes as the value of h, the vapour content.

If W is the dry portion, then

$W = \Delta - h$
= 1,176·8

Then, if 1,176·8 grammes of dry air W carry 17·3 grammes of water vapour h when saturated at 20° C., how much vapour x can 1,081·65 grammes carry? This becomes

$$x = \frac{ha}{W}$$

$\log x = \log h + \log a - \log W$
x = 15·901 grammes.

Not the dry portion only of the air at the initial temperature has to be warmed or cooled to a temperature of 33° C., but also the water vapour. The heat required for this is calculated by a method parallel to that used for calculating the result for the dry portion, but by means of the constant 0·4 for the specific heat, instead of 0·2375. There is, however, another important alteration—namely, that, unlike the dry air, the weight of vapour changes for each change of temperature, and the vapour weight has to be found by calculation before we can begin to calculate the heat absorbed in warming it to 33° C. This is done in the way exemplified in the immediately preceding paragraph, where we found that at 20° C. the vapour weight carried by 1,081·65 grammes of dry air was 15·901 grammes.

Therefore we continue the calculation for the stated temperature of 20° C. as follows:

Let n be the weight of water vapour (15·901 grammes) carried by 1,081·65 grammes of dry air when saturated at the initial temperature.
Let s be the specific heat (0·4) of water vapour at atmospheric temperatures.
Let t be the number of degrees Centigrade (13) of temperature traversed between initial temperature and 33° C.
Let C_{II} be the heat required, in large calories.

Then $C_{II} = nst$
$\log C_{II} = \log n + \log s + \log t$
C_{II} = 0·082686 large calorie.

We have found that 1,081·65 grammes, or 1·08165 kilograms, of dry air carry 35·188 grammes of water vapour when saturated at 33° C.

Our calculations show also that at other temperatures 1,081·65 grammes of dry air go with other weights of water vapour, ascertained for every given temperature. To find

how much vapour has to be added, we subtract the weight, found for the given temperature, from 35·188. Obviously this added water is derived from the respiratory passages by vaporization at 33° C., and the latent heat of vaporization is derived from the same source.

Let C_{III} be the heat of vaporization of water vapour added in respiratory passages.

Let m be the weight of water derived from respiratory passages.

Let λ be the latent heat of vaporization at 33° C. in large calories per gramme (0·58).

Then $C_{III} = m\lambda$.

At 20° C. we have 1·08165 kilograms of vapour-free air to consider first. This has with it all the water necessary to saturate it at that temperature. It is taken in with the breath, and within the respiratory passages its temperature is raised to 33° C.; its vapour-carrying capacity is thereby increased from 15·901 to 35·188. Therefore, in this case

$$m = 35{\cdot}188 - 15{\cdot}901 = 19{\cdot}286$$

and $C_{III} = m\lambda = 11{\cdot}1864$ large calories.

If, instead of 20° C., the temperature stood above 33° C., the method of calculation would be the same, but C_{III} would bear a negative sign, because water would have to be condensed instead of vaporized.

All the necessary calculations in regard to air breathed in at 20° C. have now been completed, and the method of working has been shown. For the construction of Table I, computations of the same sort have been made from - 50° C. to + 55° C. (- 58° F. to + 131° F.).

The procedure to be followed to solve the problem of varying humidities has already been pointed out—that is, the air inspired is considered for purposes of calculation as divided into two parts, one part 100% saturated, the other part absolutely dry. If in a cubic metre of inspired air the degree of humidity is 40, then we calculate 40% of the weight of that cubic metre as fully saturated, and the 60% left is calculated as absolutely dry. This method removes all difficulties, provided the table is available for reference.

Interpretation of Table I.

The table given hereunder has been constructed by the methods shown in the preceding explanatory matter, and more particularly in the sample calculation for a temperature of 20° C., chosen for illustration.

The table is set out with five headings. The first indicates the temperature to which the figures correspond. Then come four sets (only three columns printed in vertical line) of calculated figures under the symbols *a*, *b*, *c*, *d*.

The second column gives the large calories which must be added to warm, or subtracted to cool, 1,081·65 grammes of vapour-free air to a temperature of 33° C. These 1,081·65 grammes are the weight of the vapour-free fraction of one cubic metre of respired air when measured at a temperature of 33° C. and pressure of 1,000 millibars. The figures are symbolized as *a*, and the value of *a* is given for each temperature. If the values for *a* are examined, it will be seen that the figures change by a regular difference of 0·2569. The zero, or pivotal point, is at 33° C., which is 306° on the absolute scale of temperature. We find, therefore, that the whole column can be constructed from the following formula, where *a* is the figure required and T is the given temperature on the absolute scale:

$$a = 0{\cdot}2569(306 - T)$$

Thus if the temperature for *a* is 40° C., it corresponds to 313° on the absolute scale, and 306 - 313 = -7. The product of 0·2569 and -7 is -1·798, which is the figure required.

The third column gives, for temperatures below 33° C., the large calories that would be absorbed in the saturation at 33° C. of the same weight of vapour-free air (1,081·65 grammes) that was already at saturation point when inspired, but which would be able to take up more vapour when warmed to 33° C. For temperatures above 33° C. the figures represent the calories of latent heat of vapour that would have to be discharged when the same dry weight of air, saturated at the higher temperature, had to condense its water content to correspond to the weight that can be carried as vapour at 33° C. The figures in this column are symbolized as *b*, and the value of *b* is given for each temperature.

TABLE I.

Key Table for Calculation of Heat Dissipated by Respiration.

(Column 4: Constant Value of $c = 20{\cdot}409$ for all Temperatures.)

1 Temperature. (Degrees Centigrade.)	2 Values of *a*.	3 Values of *b*.	5 Values of *d*.
−50°	21·324	20·407	0·0008
−40°	18·752	20·359	0·0025
−30°	16·183	20·261	0·0064
−25°	14·899	20·163	0·0099
−24°	14·641	20·136	0·010
−23°	14·384	20·108	0·012
−22°	14·127	20·077	0·013
−21°	13·870	20·043	0·014
−20°	13·614	20·007	0·015
−19°	13·357	20·966	0·016
−18°	13·101	19·922	0·017
−17°	12·843	19·874	0·018
−16°	12·587	19·821	0·020
−15°	12·330	19·764	0·021
−14°	12·073	19·703	0·023
−13°	11·816	19·664	0·025
−12°	11·559	19·563	0·026
−11°	11·302	19·483	0·028
−10°	11·045	19·398	0·030
−9°	10·780	19·305	0·032
−8°	10·532	19·204	0·034
−7°	10·275	19·096	0·036
−6°	10·018	18·977	0·039
−5°	9·761	18·850	0·041
−4°	9·505	18·712	0·043
−3°	9·248	18·564	0·046
−2°	8·991	18·404	0·048
−1°	8·734	18·330	0·051
0°	8·477	18·043	0·054
1°	8·220	17·869	0·056
2°	7·963	17·673	0·058
3°	7·706	17·469	0·061
4°	7·450	17·253	0·063
5°	7·193	17·018	0·066
6°	6·936	16·775	0·068
7°	6·679	16·514	0·070
8°	6·422	16·234	0·072
9°	6·165	15·937	0·074
10°	5·908	15·622	0·076
11°	5·651	15·287	0·078
12°	5·395	14·933	0·079
13°	5·138	14·553	0·081
14°	4·881	14·153	0·082
15°	4·624	13·727	0·083
16°	4·367	13·276	0·084
17°	4·110	12·798	0·084
18°	3·853	12·292	0·084
19°	3·596	11·755	0·084
20°	3·339	11·186	0·083
21°	3·083	10·584	0·081
22°	2·826	9·947	0·079
23°	2·569	9·272	0·077
24°	2·312	8·560	0·073
25°	2·055	7·806	0·070
26°	1·798	7·008	0·065
27°	1·541	6·173	0·059
28°	1·284	5·291	0·052
29°	1·027	4·331	0·044
30°	0·771	3·336	0·035
31°	0·514	2·284	0·025
32°	0·257	1·163	0·013
33°	0·0	0·0	0·0
34°	−0·257	−1·240	−0·015
35°	−0·514	−2·546	−0·032
36°	−0·771	−3·930	−0·050
37°	−1·027	−5·385	−0·071
38°	−1·284	−6·926	−0·094
39°	−1·541	−8·541	−0·120
40°	−1·798	−10·269	−0·148
41°	−2·055	−12·038	−0·179
42°	−2·312	−13·998	−0·214
43°	−2·569	−16·018	−0·251
44°	−2·826	−18·155	−0·286
45°	−3·083	−20·361	−0·337
46°	−3·339	−22·770	−0·387
47°	−3·596	−25·254	−0·441
48°	−3·853	−27·972	−0·500
49°	−4·110	−30·745	−0·564
50°	−4·367	−33·678	−0·634
51°	−4·624	−36·923	−0·712
52°	−4·881	−40·244	−0·795
53°	−5·138	−43·951	−0·888
54°	−5·395	−47·517	−0·984
55°	−5·651	−51·894	−1·091

What should be the fourth column has been omitted. It gives the large calories representing the latent heat of vaporization that will be absorbed in the saturation at 33° C. of the 1,081·65 grammes which constitute the vapour-free fraction of one cubic metre of respired air, if inspired as absolutely dry air at the stated temperature. The figures do not change: they are the same for all initial temperatures; they are symbolized as *c* and must be considered in any calculation made from the table.

The last column (which, as has been pointed out, should really be the fifth) gives the heat, in large calories, which must be added to warm, or subtracted to cool, the inspired vapour of saturated air to 33° C., the temperature at which it is expired. The figures in this column are symbolized as *d*.

Note also in the table that there is a negative sign before the figures which indicate a reversal of direction of transfer of heat, causing calories to pass from the air to the body. Note once more that the calories of *c* never change their sign.

The d Column may be Neglected.

As a note at the conclusion of Table I, let it be said that though the figures for the *d*, or fifth, column had to be calculated and included, they can be neglected. The reason is because they are so small as to make no appreciable difference, and further, the order of accuracy of the fundamental measurements is not so great as to allow one to be certainly accurate about figures of the magnitude of *d*. Still, the figures are there, if wanted for any purpose.

Calculation of the Weight of Water Vaporized.

It was stated at the beginning of this paper that Table I would make it easy to calculate not only the heat, but also the moisture lost from the body by respiration. For since the latent heat of vaporization of one gramme of water at 33° C. has been taken as 0·58 of a large calorie, the vaporization heat divided by 0·58 will give the weight of water vaporized per cubic metre at the given temperature. If the respiratory ventilation current is known, we can then calculate the water vaporized per minute or per hour by respiration, under the conditions given. But an exception must be made when the air is foggy, as will be presently explained.

Respiration of Foggy Air.

The water held by foggy air is partly in the state of vapour and partly in the liquid state, suspended in fine droplets. If the air is sufficiently warmed, these droplets will be vaporized. When vaporization occurs, heat is absorbed at the rate of 0·58 of a large calorie per gramme of water. If the foggy air is inhaled, vaporization will occur within the respiratory passages. What will happen is that a certain amount of water that would otherwise have been discharged through the walls into the respiratory passages is retained in the body, its weight being equal to the weight of liquid water suspended in the fog. But the calorie discharge is not altered; it is exactly the same, whatever the source of the water that is vaporized. We may put it that the water suspended in fog spares an equal weight of water from being lost to the body by respiration.

Use in Calorimeter Estimations.

The large respiration calorimeters available in certain laboratories make it possible to measure the total calories lost from the body, and at the same time they tell what proportion of the loss is due to evaporation. It is more difficult to estimate separately the amount of heat lost by various channels. But if the total heat lost from the body by evaporation is already known, and if, as by the present means, we can calculate separately the heat lost by vaporization in the breath, the problem becomes simpler, because the other part of the water vaporized must have come through the skin surface, either as insensible perspiration or as sweat. For the rest of the heat lost, that due to voiding of urine and fæces is easily measured. In regard to loss by conduction of heat, we know already and separately the amount lost by warming of the inspired air. The rest of the heat lost escapes by radiation and conduction from the body surface.

Examples of Practical Application of Table I.

Example I.

At Sydney, at 3 p.m. on February 22, 1930, the temperature was 40·39° C. (104·7° F.), humidity 25%, and pressure 1,000·4 millibars (29·545 inches). What was the heat loss from the body per cubic metre respired?

From the table (Table I), by finding the values of *a*, *b*, and *c* at 40° and 41° and interpolating to find the values at 40·39°, we get the following:

$a = -1{\cdot}898$
$b = -10{\cdot}959$
$c = 20{\cdot}409$

The air inhaled has to be cooled to 33° C. As regards cooling, *d* is neglected and *a* is used. The figure obtained as the value of *a* is −1·898. This means that to cool 1,081·65 grammes of air from 40·39° to 33° C. (at which temperature it measures a cubic metre at 1,000 millibars), it is necessary to remove 1·898 large calories.

If we had to devaporize an atmosphere completely saturated at 40·39° C. to the content of water vapour that a cubic metre can hold at 33° C., it would be necessary to discharge 10·959 large calories. These calories, therefore, bear the negative sign. Since the humidity of the sample is 25%, we have to concern ourselves mathematically with the devaporization of 25% of a cubic metre. Therefore the heat to be discharged will be

$$\frac{-10{\cdot}959 \times 25}{100} = -2{\cdot}739 \text{ large calories.}$$

The moiety to be calculated as dry at 33° C. is 75%. The vaporization of 100% would absorb 20·409 calories, which is the value of *c*, whatever the initial temperature. Therefore for 75% it will be

$$\frac{20{\cdot}409 \times 75}{100} = 15{\cdot}307 \text{ large calories.}$$

These 15·307 calories, being all derived from the body and absorbed by the air, bear a positive sign.

The algebraic sum of the vaporization calories, therefore, is

$$15{\cdot}307 - 2{\cdot}739 = 12{\cdot}568$$

We have already found the heat loss (gain to the body, loss from the air) involved by the cooling of the air to 33° C. This was −1·898 calories. The total heat loss is the algebraic sum of the calories lost (or gained) by conduction (here −1·898) and the calories lost by vaporization (here 12·568). Thus 12·568 − 1·898 = 10·670 large calories = total heat loss per cubic metre respired at 1,000 millibars pressure.

In this particular case the observed pressure was 1,000·4 millibars, involving a correction too small to make an appreciable difference, so it is neglected.

Example II.

At Katoomba, New South Wales, at 9 a.m. on May 19, 1920, the temperature was 2·83° C. (36·5° F.), humidity 75%, pressure 909·23 millibars (26·85 inches). The height above sea level is 1,017 metres (3,336 feet).

(*a*) How much heat is lost per cubic metre respired?

(*b*) If a man in this atmosphere is respiring 24·8 litres per minute, how much heat is dissipated per minute by this channel?

(*c*) If the Katoomba figures are taken as representing a 24-four hour mean, and a man working, eating, and sleeping in that atmosphere respires 22 cubic metres in twenty-four hours, how many calories per twenty-four hours does he lose by respiration?

By reference to Table I, and interpolating as required between the figures for temperatures 2° C. and 3° C., we find that at 2·83° C. the value of *a* is 7·749 calories, *b* is 17·503 calories and *c* is 20·409 calories. We neglect *d*.

Having found the value of *a*, we have found the calories required to raise the temperature from 2·83° C. to 33° C. at 1,000 millibars pressure.

The humidity being 75%, we calculate 75% as *b*, and 25% as *c*.

Therefore $\frac{b \times 75}{100}$ = the calories required to saturate at 33° C. the moiety of air calculated as saturated at 2·83° C. at 1,000 millibars pressure. That is:

$$\frac{17{\cdot}50 \times 75}{100} = 13{\cdot}128 \text{ calories.}$$

The calories required to saturate at 33° C. the moiety of air calculated as dry at 2·83° C. and at 1,000 millibars pressure $= \frac{c \times 25}{100} = \frac{20{\cdot}409 \times 25}{100} = 5{\cdot}102$ calories.

To saturate a cubic metre there is absorbed

$$\frac{b \times 75}{100} + \frac{c \times 25}{100} = 13{\cdot}128 + 5{\cdot}102 = 18{\cdot}230 \text{ calories.}$$

Total to warm and moisten at 1,000 millibars:

$$18{\cdot}230 + 7{\cdot}749 = 25{\cdot}979 \text{ calories.}$$

The observed pressure was 909·23 millibars, and the result has to be corrected to that pressure. It is therefore

$$\frac{25{\cdot}979 \times 909{\cdot}23}{1{,}000} = 25{\cdot}979 \times 0{\cdot}90923 = 23{\cdot}621 \text{ calories.}$$

The question as to the heat dissipated per minute by a man respiring 24·8 litres per minute is easily solved, for 24·8 litres = 0·0248 of a cubic metre, and the answer is 23·621 × 0·0248 = 0·586 large calorie per minute.

The third question is answered just as easily, for 23·621 × 22 = 519·6 calories, the heat lost in twenty-four hours.

If we wish to know the weight of water vaporized by respiration per cubic metre of the same atmosphere, we find the calories required to saturate a cubic metre under the conditions existing, that is, at the given temperature and humidity; then we correct the figures to the observed pressure and divide by 0·58. The calculation is

$$\frac{18{\cdot}230 \times 0{\cdot}90923}{0{\cdot}58} = 28{\cdot}64 \text{ grammes of water vaporized per cubic metre respired.}$$

Example III.

We next consider and compare mean summer and winter records at various places, as regards their effect on respiratory heat loss. The records for these places were supplied by courtesy of the Commonwealth Meteorological Bureau. Since the method of working has already been sufficiently exemplified, details of calculation do not need to be shown again. The original data are set out in Table II, and the calculated results in Table III. In these tables, summer comprises the months of June, July, August in the northern hemisphere, and December, January, February in the southern hemisphere. For winter, it is the opposite way.

Looking at Table III, it will surprise some to find that everywhere, both in summer and winter, the heat lost by adding vapour to the inspired air is from two to three times as great as that required to warm it to body temperature, and this applies even in the coldest winter climate, as at Chicago. The present author can look back to his calculations of over twenty years ago, when he found the same thing, the occasion being a study of claims made by anæsthetists on behalf of "warmed ether".[1] In the ice-cold ether bottle the air pumped in was so cooled, right at the start, as to deposit nearly all its moisture. The correspondingly increased demand for vaporization of water from the respiratory passages removed more than twice as much heat as could be gained by warming the dried air-ether mixture. Plain, undried room air would have saved all the fuss, and twice the heat.

The figures in Table III register a heat loss that is independent of wind action. The only wind effect is the respiratory ventilation current, regulated by the body itself. The volume of air breathed per twenty-four hour period will vary with the activities of the individual and with the height above sea level—that is, with the barometric pressure. At the Katoomba altitude (see above) it is about 10% more than at sea level. The total volume breathed per whole twenty-four hours by a man doing heavy work for eight hours of the period may reach twenty-four cubic metres at sea level, but a large proportion of an average population will breathe from 15 to 20 cubic metres per twenty-four hours. If a man breathing the winter air of Chicago (see Table III) loses 9·174 calories per cubic metre by warming the respired air, and 19·219 by moistening it, he will by breathing 20 cubic metres per day lose 183·5 by warming and 384·3

TABLE II.

Summer and Winter Mean Temperature, Humidity, and Pressure for various Places.

Place.	Temperature. (Degrees Centigrade.)		Humidity. (Percentage.)		Pressure. (Millibars.)	
	Summer.	Winter.	Summer.	Winter.	Summer.	Winter.
London	16·2	4·31	72·0	85·66	1015·46	1015·46
Chicago	21·94	−2·05	72·0	77·3	1015·24	1018·85
New York	21·86	−0·66	72·6	64·3	1015·8	1017·83
Sydney	21·6	12·36	67·0	74·6	1013·09	1017·83
Melbourne	19·22	10·01	59·6	80·4	1013·09	1018·17
Brisbane	24·77	15·44	65·0	71·0	1011·74	1018·50
Capetown	20·94	12·96	66·0	86·6	1013·43	1015·24

TABLE III.

Heat Loss in Calories per Cubic Metre Breathed, showing separately the Losses due to Warming and to Saturating the Air, under the Climatic Conditions set out in Table II.

Place.	To Warm.		To Saturate with Vapour.		Total Summer-Winter Difference.
	Summer.	Winter.	Summer.	Winter.	
London	4·383	7·482	15·439	17·908	5·568
Chicago	2·885	9·174	13·1	19·219	12·408
New York	2·907	8·8	13·091	18·993	11·795
Sydney	2·967	5·397	13·757	16·237	4·92
Melbourne	3·586	6·013	14·893	16·849	4·383
Brisbane	2·14	4·594	12·473	13·772	3·753
Capetown	3·14	5·227	13·044	15·573	4·616

by moistening, totalling 568. In the Brisbane winter climate, the corresponding figures per cubic metre are 4·594 by warming and 13·772 by moistening, and for 20 cubic metres they come to 91·9 and 275 respectively, with a total of 367. Thus for 20 cubic metres breathed, the Chicago man loses 92 more by warming the air and 109 more by moistening it than he would in Brisbane. The difference in total calories comes to 201 per 20 cubic metres breathed. If other things were equal, heat production would have to be "stepped up" in the Chicago man to cover the difference. But if the balance, or part of it, were compensated by a decrease in heat loss through the skin, then metabolism would be to that extent unaffected by the climatic difference.

Conclusion.

As regards metabolism, the reference in this paper is confined to air respired. But, of course, the air respired is only one factor. The temperature, humidity, velocity, and pressure of the air, and the extent of radiation, as they operate in the room and beneath the clothing, are factors influencing metabolism through the skin surface. Of these, velocity of air movement and radiation, both of extreme potency on the outer surface, are excluded in respiration.

The remarks just made must apply in their degree to the measurement of basal metabolism in different places and circumstances, but no more can be said about it here.

Summary.

The mode by which heat is exchanged between the body and the air during respiration has been studied with a view to measurement of the quantity of heat transferred.

A method has been devised and explained by which the measurement can be made in calories when the following data are available: the respiratory ventilation current, the temperature, the humidity and the atmospheric pressure.

In order to make it easy to calculate the heat loss for any temperature, humidity, and pressure, a key table (Table I) has been constructed covering a range from −50° C. to +55° C. (−58° F. to +131° F.). Examples are given to illustrate the method of calculation from the table.

With this table the heat loss can be calculated as per cubic metre respired, or per minute, or per twenty-four hours. Or it may be calculated as for the mean temperature, humidity, and pressure at the different seasons of the year at different places—London, Chicago, New York, or anywhere. Examples of this method of study are given, and various places are compared.

It is also possible to use the table for calculation of the water vaporized in the respiratory passages per cubic metre respired. An example of such a calculation is given.

Reference.

(1) C. E. Corlette: "The Heat Losses of the Body Connected with Surgical Operations under Ether Anæsthesia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, August 6, 1921, page 99, and August 13, 1921, page 115. (Also subsequent correspondence.)

THE ORGANIZATION OF A CASUALTY CLEARING HOSPITAL FOR AIR RAIDS.[1]

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IN the army, not only must the actual treatment of the wounded be considered, but also their evacuation from the front line. This applies also to air raid work amongst the civilian population. Consequently, equally as important as the treatment of actual air raid casualties is their evacuation from hospitals in a vulnerable area to those in a less vulnerable area.

One of the most important links in the chain of evacuation is the hospital corresponding to an army casualty clearing station. I was stationed in a small London voluntary hospital of 100 beds, situated in an area subject to almost continuous air raids. This hospital fulfilled the function of a casualty clearing station, and it is this type of institution with which I shall deal.

COORDINATION OF HOSPITALS.

In order that medical services may function adequately for the reception, treatment and evacuation of air raid casualties, each hospital must function as a unit in a well-coordinated scheme. No hospital can function as an independent unit except temporarily, when it may be isolated from its fellows by breakdown of communications.

For this purpose of coordination London was represented by a large circle, with its centre approximating the position of the city of London itself. This circle was divided into several sectors, each containing many hospitals of different types. The general scheme of evacuation was from the centre outwards. Near the centre were large teaching hospitals, one to each sector, and a senior member of the visiting staff was usually appointed the sector head to control every hospital in his area. He also had control of the ambulance services of that area, which themselves were under their own administrators. He himself was under the direction of the Ministry of Health.

These large teaching hospitals, by virtue of their vulnerability, were not able to be used to their full extent, and actually acted more as main dressing stations. That this was a wise policy is shown by the number of these hospitals which suffered during the air raids.

Further out from the centre, in places like the East End, were the many small voluntary hospitals and London County Council institutions which were used as casualty clearing stations. London is fortunate in that she has many small hospitals, and that city which has many small scattered hospitals connected with each other by many routes is far better off than that which has only a few large hospitals. In the former, if a hospital is put out of commission for any reason (for example, through having received hits from bombs), then it can be by-passed and patients sent to other hospitals near by. Again, a group of these hospitals may act like a group of casualty clearing stations. When one is full it closes down and further patients go to another hospital. In a city with only a few large hospitals the destruction of one may seriously impede medical services.

Further out still, well into the country, were placed large base hospitals. These were either large county hospitals or specially built hut hospitals. They themselves were served by convalescent institutions.

A scheme such as that described above works very well; but one must remember that aerial warfare may upset all calculations and the bases may be the first to be wiped out.

THE CASUALTY CLEARING HOSPITAL.

The best type of casualty clearing hospital suited for work in vulnerable areas should consist normally of 100 beds, capable of expansion to 200 beds in an emergency. It should have its own radiographic and out-patient departments and auxiliary services.

The Staff.

In peace-time it will probably be run by a secretary-superintendent under the control of a lay board. The medical staff consists of a group of visiting specialists assisted by a house team under the control of the resident surgical officer.

When air raids commence this staff must be considerably changed and may be discussed under the following headings: (i) the medical superintendent, (ii) the medical staff, (iii) the nursing staff, (iv) the lay staff.

[1] A post-graduate lecture, delivered under the auspices of the New South Wales Post-Graduate Committee in Medicine on June 22, 1942.

The Medical Superintendent.

The medical superintendent, usually a member of the honorary staff, lives in the hospital and is directly answerable to his sector head. His work is comparable with that of the commanding officer of a unit, ensuring the smooth running of all parts of the hospital. He works in close liaison with the secretary and his board, who represent the "Q" and ordnance side of the institution.

Many may ask whether any consultant would willingly give up all his practice and become again a resident medical officer at a salary. It must be remembered that when air raids commence consultant practices disappear overnight.

The Medical Staff.

The medical staff itself consists of eight units. The first is the senior air raid surgeon, who should be of senior consultant rank, though he may not necessarily have been on the same hospital staff before the raids began. It was originally thought that he should come to his post only when called upon; but this is not practicable, because during a long air raid one cannot cross the city without incurring needless risk from bombs and anti-aircraft fire. Also roads may be blasted and your consultant is stranded, useless. He therefore sleeps in hospital, even if he is not present during the day. The senior surgeon is responsible for the surgical work of the hospital during raids, and he in turn delegates a great deal of his work to his junior surgeon, as he cannot work for twenty-four hours without rest. Again, when a rush of casualties occurs, two operating theatres will need to be used.

The junior surgeon must be a man of good surgical experience, and in England he was recruited from the ranks of surgical registrars and resident surgical officers. His duty is to reside permanently at the hospital.

It is well that both surgeons have a good knowledge of general surgery, and especially of orthopædic surgery, because 90% of air raid surgery is orthopædic in nature.

The number of surgeons needed by an individual hospital will depend on its size and its position in regard to vulnerable areas, so, in addition to the surgeons allotted in the above manner, the sector head will send out mobile surgical units, consisting of a surgeon, his assistant, an anæsthetist and an instrument sister. They have their own transport and are fully equipped with instruments, dressings and a small operating table. These units travel to hospitals whose surgeons may be temporarily unable to cope with a sudden overwhelming number of urgent surgical patients.

The air raid physician is a man of consultant rank. He assists in the examination of the wounded, in post-operative treatment, in the care of chest casualties, in the treatment of shock and burns, and so on. He is especially important when gas casualties occur, and is the leader of the gas decontamination squad. In addition, when required, he acts as an anæsthetist.

The senior anæsthetist is usually a specialist who should also live at the hospital. He is responsible for the anæsthetic work, particularly in the difficult or complicated cases, and is assisted when necessary by the physician and the "housemen", all of whom should be proficient in giving ether satisfactorily.

In addition, each sector has consultant ear, nose and throat, eye, skin and mental specialists, who travel where they are needed. These are apart from those men attached to the special hospitals.

The "housemen", of both sexes, should all have had good general hospital experience and be prepared to fill any gap. All should be able to act as relieving anæsthetists or operating theatre assistants, and be trained in grouping and typing blood.

To one is delegated the task of being resuscitation officer, skilled in giving intravenous injections. He is in charge of the blood and serum bank. Any "houseman" should, however, be able to take over if necessary.

To a senior "houseman" is delegated the work of receiving officer. His special job is to examine all patients on their arrival, inspect and remove tourniquets, decide whether resuscitation is necessary, and roughly diagnose the patient's injuries. He should not be expected to make a perfect diagnosis except in continuous hæmorrhage and sucking chest wounds, because air raid casualties, when admitted to hospital, are filthy in the extreme; their condition renders diagnosis most difficult, and most of these patients cannot be cleansed until they have been resuscitated.

Another "houseman" (or more if they are available) is detailed to each ward to look after the patients and carry out the instructions of the surgeons.

In London the senior medical students were sent out from the teaching schools and allotted to various hospitals, where they became resident students and were given an invaluable training in the surgery of the injured. Apart from gaining wonderful experience, these men fulfil a very good purpose, in that they can assist in resuscitation work, act as clinical clerks, and generally aid where necessary. (For example, they may have to act as stretcher bearers.) They are also trained in gas decontamination work and the work of the receiving room. They act as operating theatre assistants and anæsthetists.

Such pathological work as is required in this type of hospital may be carried out by a good technician. The services of the specialist are utilized at the bases or in special laboratories.

The X-ray work can be done by a well-trained radiographer, and in London we had women radiographers. They are supervised by a consultant, who is usually attached to several hospitals. A portable machine is the handiest apparatus.

The Nursing Staff.

The matron and her assistant matron are in close liaison with the superintendent of the medical staff. The matron has under her care the normal nursing staff, which must be reinforced with extra nurses, either recalled back to the service or brought in from other hospitals not sustaining the brunt of air raids. In addition, we may have women specially trained for air raid work brought in to work only until the war is ended.

We must remember that during raids there must be as large a staff on duty at night as in the day, so that almost double the normal number of nurses is required. This applies especially to special staffs, such as operating theatre staffs, because of the incessant night work entailed.

In addition to the nursing staff there are the Voluntary Aids and the Red Cross workers. The former can help a great deal in the wards and lighten the nurses' tasks. One important function of the Red Cross workers is the running of the canteen service, placed in the receiving room, for the ambulance drivers and hospital staff and patients. This canteen is run day and night.

The Lay Staff.

I have already mentioned the secretary, and with him are the almoner and her staff. These people fulfil a most important duty, especially in the receiving ward. Each patient on admission to hospital should be accompanied by a card, not unlike the army field medical card, attached to his clothes. The almoner and her staff collect the information on this card into the equivalent of an admission and discharge book under instructions from the receiving medical man, and they attach any directions to the patients on separate slips—for example, "to medical ward", "to surgical ward" *et cetera*. They also collect any clothes or valuables removed from casualties, either in the receiving room or in the wards, and label and store them.

In addition, the almoner has an important task in finding out from the local authorities whether patients' homes are still standing. Many patients are admitted to hospital suffering from mild shock and little else, and may soon be discharged; but if they have no homes to which to go, they can be discharged only when the local authority can make provision for them.

Other clerks, such as book-keepers, stenographers *et cetera*, are invaluable in the wards, doing rounds with the medical officers, writing down their remarks and keeping

records. Where necessary, too, they can assist in different departments—for example, in the gas decontamination chamber, in extinguishing incendiary bombs or in stretcher bearing.

In the receiving room are placed one or two clerks, whose duty it is to look after the stretcher dump and exchange of stretchers, blankets and Thomas splints received from the ambulances.

The kitchen staff, under the control of the housekeeper, is most important and must be able to serve hot meals and liquids at any hour of the day or night. Therefore this staff must be reinforced during air raids.

The auxiliary staff consists of the porters and wardsmen, who are all instructed in first aid and in gas decontamination. They act as stretcher bearers as well and unload ambulances. In addition, from various organizations, such as the Boy Scouts, are recruited runners to act when the telephones fail, as they are bound to do, even if only temporarily. These lads use motor bicycles or bicycles, and are most useful. They also watch for fire bombs and deal with them when the need arises.

Hospital Arrangements to be Made Prior to Air Raids.

All air raid plans of the hospital, including the staffing as already described, must be made long before aid raids commence.

These plans may be grouped under the following headings: (i) adequate beds, (ii) additional stores, (iii) air raid protection of the buildings, (iv) protection of the staff and patients, (v) arrangements of the wards, (vi) duplication of services, (vii) provision of a blood bank, (viii) provision for gassed patients.

On the threat of air raids all unnecessary hospital work in vulnerable areas should cease. Urgent surgical and medical patients must of course be dealt with.

Each hospital should have a base and convalescent depot, adequately staffed and equipped, to which all patients can be evacuated at regular intervals.

Patients suffering from chronic disease (for example, those requiring surgical treatment, but not urgently) are sent to the base, as also are convalescent "urgent surgical" patients. In other words, a hospital in a vulnerable area must be regarded as a casualty clearing station and no more.

In London a 100-bed hospital was given a further 100 beds with bedding and blankets by the Ministry of Health. These were treated as war equipment stores and used only when raids commenced.

Additional stores are laid in, especially dressings, plaster of Paris, bandages in airtight and watertight containers, extra tinned food, and so on. Extra anæsthetic material (for example, ether) is stored, and owing to its dangerous nature special provision must be made for its storage. It is necessary to construct a small concrete store-room well away from the main hospital, preferably underground, in order to obviate any explosion that may occur.

Air raid protection of hospitals is a matter for the architects. All unnecessary glass must be removed, especially from operating theatres, and replaced by bricks or boarded up. Doorways must be protected by thick brick walls. Sandbags do not last long enough and are a temporary expedient. All blackout precautions must be taken so that work is not impeded by blackout conditions.

With regard to air raid protection of the staff, it may be stated that some refugee doctors, who toured German clinics as far back as 1935, informed me that they were impressed with the underground wards and operating theatres, specially constructed of concrete, for use in air raids. Unfortunately we in London had none of these.

In hospitals one has to protect three classes of people: (*a*) hospital in-patients, (*b*) members of the staff on duty during the raids, (*c*) those who should be out of the way, either resting personnel or people such as out-patients.

Without adequate underground shelters and wards the protection of in-patients is most difficult and reduces one to the expedient of keeping all top wards empty; this means that a 200-bed hospital may be promptly reduced to 100 beds. Those patients who can walk go to shelters, and those who cannot are placed under the beds whenever possible and away from glass windows.

Previous ideas of hit-and-run raids, in which everyone goes to the shelters for a short while, are of course no longer tenable. In prolonged air raids there must be personnel working who cannot go to shelters. They must be supplied with gas masks and tin helmets. Should gas be used, gas suits must also be available. The protection of the hospital is one of the best means of protecting the personnel.

In prolonged raids we must do better than slit trenches. Where out-patient departments fuction (and not even raids dim the ardour of some out-patients) there must be adequate shelters near the hospital for these people.

Deep shelters are not, as has been said, refuges for cowards, but places of protection and rest for those whose services are not at that moment required; for example, they are shelters and resting places for the staff off duty, and must be provided with adequate bunking space and sanitation. In case of nightly raids the members of the day staff go straight to shelters on the alarm and sleep there until morning. They are then protected not only against bombs, but also against noise, which is one of the causes of loss of sleep so detrimental to good work. This last statement refers also to factory workers.

Arrangements of the Wards.

The following types of wards are required; and incidentally we must remember that in civilian work casualties are male and female, so that every ward must be duplicated.

The receiving room must be situated so that it is easy of approach by ambulances and of easy access to various wards. It should be placed in the out-patient department and should either be on the same ground level as the wards or, if it is on a different level, there must be an incline joining them, so that no stairs have to be used. Lifts must not be relied upon, owing to the probable cutting off of power.

There must be a separate entrance to and exit from the receiving room, so that there is no need to turn motor cars. This drive should be off a side road and not a main road. The entrance, as all entrances, must be on the "double lock" principle, so that doors can be opened without a stream of light running across the footpath.

The receiving room is divided into three main sections: (*a*) A large section for the reception and examination of patients; this may be partitioned into two for the different sexes; it has a number of low tables on which patients on stretchers may be placed, to obviate the necessity of examining patients on the floor. (*b*) A canteen for refreshments for the ambulance drivers and staff and hot fluids for the patients; hot coffee and tea should be ready to be served at any time. (*c*) A stretcher, blanket and Thomas splint dump presided over by clerks, so that the ambulances can leave their loaded stretchers and go away with empty ones without delay.

The resuscitation ward is partitioned for male and female patients; it is best placed at the end of a corridor, so that it will not be converted into a thoroughfare. It is equipped with all the apparatus for the treatment of shock, and next to it is a room holding the blood bank and means of sterilizing transfusion sets.

It is an excellent idea to have small separate wards for cleansing the bomb casualties who need operative treatment. Bomb casualties on admission to hospital are in an indescribably filthy state; eyes, ears, nose and mouth are blocked with dirt and dust *et cetera*. This cleansing may be part of the operation and may have to be delayed until the patient is anæsthetized.

Other wards required are (i) "pre-operative" wards, for preparation of casualties for the operating theatre, (ii) "post-operative" wards, (iii) observation wards for patients not requiring operation, (iv) the evacuation ward. The last mentioned fulfils a good purpose, in that here patients fit for evacuation may be sent pending the arrival of ambulances. This saves much unnecessary delay in the loading of ambulances, which may be important when raids occur at frequent intervals during the day as well as at night.

Duplication of Services.

All hospitals must have a duplicate method of cooking, heating and sterilizing, and one of the methods must be by steam worked from a boiler-house, because power and gas may possibly be cut off by explosion.

Every hospital should have a good store of large torches and batteries (lamps are dangerous), in case the electric light fails.

The operating theatre must, of course, be provided with a battery set in case the main lights fail.

The Blood Bank.

Each hospital should have its own blood and serum bank. Blood can be collected at the hospital and stored. Serum will be collected from a central manufacturing centre, as in Sydney. At these bases there are usually the necessary staff and equipment to manufacture their own serum, but this will not be possible at a casualty clearing hospital.

Each hospital therefore should have a long list of donors, from whom blood can be taken from time to time. At London we used to do this every Saturday afternoon, and the blood was refrigerated for any emergency.

Should serum be prepared at a central bureau, care must be taken to ensure adequate supplies even during raids. There should be no need to worry about donors during emergencies.

Provision for Gassed Casualties.

Prior to air raids a large concrete chamber separate from the hospital should be erected for the decontamination of mustard gas casualties. This is so partitioned as to have a small receiving room and a decontamination room, where patients are decontaminated and undressed. The contaminated stretchers are passed back to the bearers, who are dressed in gas-proof clothing. The stretchers and ambulance are decontaminated by the ambulance authorities and are no concern of the hospital.

Once decontaminated, the patients are sent to another room on fresh stretchers, where fresh clothes are donned, and they are now fit to enter the wards with non-gassed patients.

Patients, no matter how they are suffering from shock, must be decontaminated, as one contaminated gas casualty may infect a whole hospital. It may be a matter of sacrificing the one for the many. The severely wounded bombed patient who is also gassed may be almost impossible to save.

The gas personnel should be in charge of a medical man, usually the physician, trained in gas decontamination work, and he is assisted by a team of medical students (if available), nurses and porters.

Before air raids occur there should be frequent practices for all personnel, as it is surprising how many mistakes are made at first.

Procedure on the Advent of Air Raids.

At the scene of a bomb incident the injured are either dug out or picked up and then examined by a medical man, who travels with a flying squadron from one incident to another. Hæmorrhage is stopped, sucking chest wounds are covered with a large pad, Thomas splints are applied, morphine is given, and other necessary first aid work is done.

This examination and treatment may be easy enough in day-time; but at night-time, when raids usually occur, it is very difficult indeed in blackout conditions to decide whether a patient is seriously hurt or suffering slightly from shock. In any case, the latter patient, if he has been near the explosion, may be suffering from lung blast, which must be treated by immediate prophylactic admission to hospital.

That is why almost all injured must be removed to hospital for treatment in bed. Consequently there is little need for first aid posts, and if they are used they should be attached to the hospitals. In other words, there should be no delay in transporting a patient from the incident to the hospital.

A field card is attached to the patient; it should show not only the patient's apparent injuries, whether morphine was given, and whether a tourniquet was applied and the time of application, but also where the patient was found. For example, if he was found in the open, near where the bomb exploded, lung blast will be suspected later; if he was found crushed beneath débris one will carefully watch the kidneys.

Should gas have been used, the ambulance will be distinguished by a special coloured card, and it will carry none but gassed patients.

At the hospital the ambulance is unloaded by hospital bearers and the casualties are taken to the receiving room, unless gas has been used, when the ambulance goes to the gas receiving room. Ambulance drivers must never be expected to unload their ambulances, as they have enough to do without this extra physical strain.

The ambulance driver and attendant, having parked the ambulance out of the traffic lane (there should be a large parking space over the receiving room), are provided with refreshment. They are then given stretchers, blankets and Thomas splints (if used) from the dump, in exchange for the ones they have taken in, so they can leave fully equipped.

The patient, still on his stretcher, is placed on a low examination table and seen by the receiving medical officer, who inspects his field card, notes any tourniquet which may have to be released, checks hæmorrhage, or sutures gaping sucking chest wounds. He is attended by the almoner or her assistant, who takes the field card information and pins onto the patient the cards showing where he is to go.

Patients for urgent resuscitation go direct to the resuscitation ward, where the team gets to work. Decontaminated gas patients are allowed here and in the other wards.

Thence they are sent to different wards, according to what is to be done to them, for observation, operation, treatment or X-ray examination.

Patients dying in the receiving ward should be sent to the mortuary. Patients picked up dead at an incident should be sent direct to the local authority's mortuary and should not be a burden on the hospital.

Some Observations on Treatment.

The treatment of air raid casualties will not be discussed specifically except for some general notes.

Anæsthesia.

The best methods of anæsthesia are as follows.

1. Ether given by the "open" method should be the main anæsthetic agent. Naturally, ether given intratracheally may be used in certain necessary cases. Ether given by apparatus needing cylinders of oxygen may be used; but the more complicated the method, the less likelihood of success. Cylinders of oxygen may be impossible to obtain.

2. Nitrous oxide and oxygen, if procurable, is excellent, especially in those cases in which great relaxation is not required.

3. "Pentothal Sodium" given intravenously is good, especially in those cases in which lung blast is suspected. In this instance ether tends to accentuate this condition.

The Gangrenous Appendix.

In a rush of casualties one is always apt to strike our old friend the gangrenous appendix and the ruptured peptic ulcer. During the rush and bustle of air raids one tends to regard them as being interlopers; but it is to be remembered that a young workman with an acute appendicitis is just as worthy of being saved as an elderly man with a perforating abdominal wound.

Minor Injuries.

Minor or apparently minor injuries must receive the same careful attention as perforated bellies. A very small bomb fragment may have a minute entry wound.

but cause severe muscle damage and so predispose to gas gangrene. Only careful attention will save these patients from disaster.

Fractures of the Arm.

Casualties with fractures of the arm should not have aeroplane splints (plaster or otherwise) applied to them in the casualty clearing hospital, as they are very inconvenient and difficult to transport. They are best treated with the arm by the side. Aeroplane splints may be applied at the base hospital.

Fractures of the Femur.

Fractures of the femur, compound or simple, are difficult problems even apart from shock, because of the necessity for evacuation. A Thomas splint with skeletal traction through the tibial tuberosity is excellent, though strapping may be used temporarily in non-compound fractures. The extension cord should be tied to the end of the splint for travelling purposes; but while the casualty is in bed one should also apply weight extension to the end of the Thomas splint, so easing the pressure on the patient's ischial tuberosities. Hodgen's splint and Böhler's frame are of course useless here.

If the patient has some days' travelling ahead of him, a well-fitting plaster spica may be used, because a Thomas splint is apt to cause pressure sores on the buttocks; so also, of course, is a badly applied spica.

At the base, of course, when probably no further transport is necessary, the usual methods of treating fractured femurs may be used.

Burns.

In spite of all that has been said about the subject recently, there is only one treatment for burns at a hospital in a vulnerable area. First, shock is counteracted and then the burnt area is cleansed under anæsthesia and coagulated by a 5% solution of tannic acid followed by a 10% solution of silver nitrate, giving an immediate tan. I say this even for deep burns. Burns of the hand or face may be treated by thorough, gentle cleansing and the application of "Vaseline" *tulle gras*, though face burns also do well with tannic acid. This treatment saves life and pain.

At the base, if the wound is very deep or if the tan becomes infected, as it will do especially in the buttock region, one may use the saline method of preparation for later skin graft, as described by Major B. K. Rank (*The Australian and New Zealand Journal of Surgery*, Volume XI, Number 3, January, 1942, page 176).

Lung Blast.

Lung blast is now a well-defined clinical entity and must always be considered. Any patient found in the region of an exploded bomb must be suspected of suffering from lung blast and the prophylactic treatment is rest in bed. X-ray examination will reveal it before clinical signs are apparent in slight cases. There is, of course, no doubt about a severe case, in which the condition may be apparent immediately. In less severe cases signs will develop twenty-four to forty-eight hours after the explosion, and the condition is diagnosed by pain in the chest, a rise in temperature and early lung consolidation. Lung blast is the most common type of bomb blast effect on land, and we must remember that it may turn the scale against a successful operation.

Bomb Blast.

In those hospitals near the sea which receive air raid casualties from bombed vessels, blast effects in the abdomen occur more frequently than in the chest of the injured who have been "blasted" while immersed in the water. These blast effects in the abdomen vary from subperitoneal hæmorrhages to rupture of a solid or hollow viscus.

Crush Injuries.

In crush injuries a careful watch on the urine must be kept; this is especially so with regard to those patients who have sustained prolonged crushing of the limbs, as one may later find the clinical picture of a severe nephritis—hæmaturia, oliguria, anuria and uræmia. It must also be remembered that sulphapyridine may cause backache with hæmaturia, and later oliguria and even anuria.

Severe Wounds.

For patients with severe air raid wounds, such as compound fractures, one must make use of the local and general exhibition of the sulphonamide derivatives. Sulphapyridine given orally is apt to cause frequent vomiting, and should be replaced by sulphadiazine if supplies are available.

Evacuation of Patients.

Patients must be evacuated from vulnerable hospitals for many reasons.

1. Those with special injuries (for example, facio-maxillary, chest and head injuries) should go to the special centres. However, if communications are broken, they must be looked after at the casualty clearing hospital.

2. Convalescents are evacuated to release beds for further casualties.

3. One very important reason is that bombed patients are in a state of fear every time a raid occurs, so that they should be evacuated as quickly as possible to less susceptible areas.

4. Patients should be evacuated if the hospital is bombed or if services fail.

5. If a time-bomb falls near by, the whole hospital, including patients and staff, must be evacuated; this requires great cooperation from the ambulance personnel.

It must be realized, too, that personnel working continuously in severe air raids are apt to suffer from the effects of strain, overwork and lack of sleep. Consequently as soon as a member of the staff shows signs of exhaustion, he is sent back to the base to work and a member is sent forward from the base; this gives an exchange of duties and conditions, with relief to all.

Conclusion.

In conclusion I wish to reiterate my statement that to deal successfully with air raid casualties we need all our hospitals, both city and country, to work as one coordinated medical service rather than as independent units.

Summary.

A brief discussion on the medical organization required to deal with civilian casualties in an area subject to air raids has been given. The casualty clearing hospital and the part played by it in the scheme of treatment and evacuation of patients have been specially chosen for description. Some general points in the treatment of air raid casualties have been stressed.

EARLY TREATMENT OF BATTLE WOUNDS OF THE HEAD.

By Douglas Miller,

Major, Australian Army Medical Corps, Officer Commanding Neurosurgical Unit, Australian Imperial Force.

During recent campaigns in the Western Desert the writer worked first as surgeon in a forward operating team, and later as officer commanding the British "Head Centre" for the Middle East forces. The knowledge gained in some aspects of head surgery in these conditions is likely to be applicable to warfare in Australia, where it is possible that campaigns may be conducted in circumstances and surroundings resembling the Western Desert.

The fortunes and surprises of war force men from the path they may elect to follow, and while the surgeon versed only in general surgical techniques must at times

be prepared to discharge the duties of a specialist branch, the man who essays to hold aloof from general work may be of little value in the emergencies of war surgery. The following are some of the points worthy of emphasis to anyone who may be called upon to deal with head wounds.

Time of Treatment of Head Wounds.

Failure to perform primary suture and to obtain first intention healing of a head wound is a grave misfortune to the patient. Even simple scalp wounds, if they become infected, are very apt to result in necrosis of bone, extradural abscess or infective sinus thrombosis. They merit all the care of deeper wounds. The practice (now so generally and wisely adopted) of leaving battle wounds in other areas unsutured has no application in the surgery of the head, and must at almost all costs be avoided. This departure from usual practice was not generally realized, but cannot be too strongly stressed.

The alternative to scrupulous *débridement* and closure of deeper wounds is infection, which means meningitis, cerebral abscess or an infected cerebral sinus. In the case of head wounds it is fortunate that the usual time limit of ten hours set for the *débridement* of wounds in other situations does not hold. Thorough surgical treatment of head wounds may justifiably be carried out after a much longer interval from the time of reception of the wound. This longer period of safety had come to be an accepted fact in civilian casualties, in which one may count on safety for about forty-eight hours; but in wounds in the Western Desert it was found that successful excision and closure with drainage were done as late as three or four days after the wound had been inflicted. The peculiar absence of gas gangrene and other severe infections and the use of sulphanilamide are responsible for this. Possibly conditions in Northern or Central Australia may prove to be the same.

If we bear in mind also the fact, now abundantly proven, that patients stand transport very much better before an operation than after, it becomes an obvious teaching that casualties with head wounds should be given the benefit of their longer safe period, and be evacuated even for some distance to a suitable operating centre before being subjected to any active surgical measures. In such a centre it should be possible to nurse them for at least a short recovery period. Nothing is more fraught with danger than partial interference with such wounds in forward field units, from which an early and exhausting evacuation will have to be made.

The following case is illustrative of primary treatment at a late stage. I sent and had returned this "follow-up card" concerning a patient treated in a desert casualty clearing station.

Three days after injury débridement of G.S.W. head, excision of wound and bone edges. Removal of damaged brain and of a large F.B. from the occipital lobe, wound closed without drainage.

The patient was evacuated to a base hospital three days after operation. The "follow-up card" was completed at the British Head Centre some weeks later:

On admission rather drowsy, some neck stiffness, C.S.F. pressure 170 mm., slightly coloured C.S.F. Homonymous hemianopia. His wound healed by first intention, he made a rapid recovery, and is now able to walk well, though hemianopia persists. X-ray shows small pieces of metal scattered around the wound.

Several such examples could be cited in support of the plea for utilizing this longer safe period, though naturally it must be remembered that early treatment under proper conditions is more likely to be successful. Many cases could be quoted, however, to show that infection and disaster are likely to follow early treatment which is inept or incomplete.

The Operating Centre.

Counsels of perfection are unlikely to prevail in war, and the surgeon dealing with head wounds, though he cannot always be an experienced neurological surgeon, must be familiar with the techniques concerned especially in the care of damaged brain and in the skilful control of intracranial bleeding. Such things cannot be done with knowledge alone, but demand some basic equipment. The most important item is surgical suction. There is only one way to keep a head operation field clean, and that is by suction. Swabbing and pressure aggravate the trouble they aspire to stop, and may do irreparable damage. Australian Imperial Force surgical teams were equipped with a foot pump suction apparatus (perhaps exhausting to an orderly, but otherwise very satisfactory) in places where motor or water suction were not available. The sucker is used to remove blood from the field and to allow the bleeding vessel to be seen and accurately controlled. Cushing clips should be carried, as it is unlikely that electro-surgical equipment will be available. Other necessary instruments are: bulb hand syringes for washing the wounds, good bone perforating instruments and good bone forceps. Surgeons will be repaid for care in seeing that their burrs, perforators and bone-cutting forceps are sharp.

Some Special Problems.

In contradistinction to civil head injuries the majority of battle head injuries are open wounds, and most of the patients have suffered only local brain injury. In civil injuries the patient's head is usually thrown against some object, and the brain suffers as a whole; but the ma'ority of battle head injuries are due to a small, high-velocity projectile striking one part of the head and causing only local cerebral damage. Usually in such cases there is no history of loss of consciousness, and until other influences are manifest the patients remain conscious and alert. The usual story is for a man to feel a hit on the head and immediately notice the local effect, such as hæmorrhage, or the possible neurological effect, such as a hemiplegia, monoplegia or hemianopia.

A young Hussar officer was hit while in his tank. He had the sensation of seeing a piercing bright light, did not lose consciousness, but immediately became hemianopic and aphasic. He had a perforating wound of the left temporo-occipital region.

Another young trooper, while fighting in his tank during a night battle, felt a hit on his head and noticed immediately that his left arm and hand were limp and powerless. He continued to fight throughout the night and at no time lost consciousness. He had received a localized right parietal lobe injury, and some weeks later recovered completely from his monoplegia.

In all such cases, of course, the wound is the primary consideration.

Surgical Treatment of Wounds of Unconscious Patients.

In some instances patients do sustain a generalized brain injury, as evidenced by loss of consciousness, in addition to a local wound. In these cases the surgeon must decide whether or not he is to operate on the wound. It often appears almost certain that the patient will die as the result of the general head injury, and the surgeon, usually sorely pressed, begrudges the time and effort of performing an unnecessary operation. If, however, the patient survives the gross brain injury, he will then have to weather the hazards of an untreated head wound. When the time margin is available, it may well be used for observation; but when this period is drawing to an end the best practice is to treat the wound thoroughly, so that if the patient survives it will not further prejudice his recovery.

The Technique of Wound Excision.

"Pentothal sodium" is the best anæsthetic agent. It can be given satisfactorily in glucose and saline solution by the continuous drip method, or a single injection is useful during the painful shaving of the scalp or introduction of "Novocain", under the influence of which the operation may be completed.

Good barbers' scissors, hair clippers and open blade razors are as invaluable as the orderly who knows how to use them. Much time is saved by expert shaving of the head. The shaved scalp should then be swabbed tediously

for some minutes with spirit, and the wound securely draped off so as to allow the anæsthetist easy access to the patient's face without disturbance of the drapes. Thoroughness is the key word in excision of the wound. Free mobilization of scalp flaps will facilitate easy approximation over quite a wide gap. This is best done by pushing the gloved finger out under the galea. Bone edges are most easily excised by the use of sharp rongeur forceps, which as a rule can be introduced through the bone defect, or failing this, through a separate opening made for the purpose. This method is quicker, easier and as good as excision of a fracture *in toto*. Soiled and torn dura must, of course, be excised with scissors, and bleeding from the dural edge must be controlled either by silver clips or by an under-running suture. When no endotherm is to be had, the red-hot tip of a probe is useful to seal small bleeding points in the dura.

An X-ray film may be available to give information about foreign bodies. I do not think this is important, as foreign bodies lying superficially in damaged brain are likely to be readily detected, and those which do not lie to hand are best left alone, until such time as their removal may be indicated. The method of gentle irrigation and suction of pulped brain is well known. If a rather stiff rubber catheter is used, it will readily detect foreign bodies. This gentle washing and sucking and a most cautious removal of foreign bodies should be persisted in, until a clean field is achieved. It may be difficult; but the surgeon must remember that irrigation and suction are the only permissible manœuvres, and at most swabbing must be limited to the placing of a small moist wool swab on a bleeding point. Hæmorrhage from most of such bleeding points will stop after a little warm lavage or may be controlled by a Cushing clip or a small suture. Attempts to use artery forceps will be followed by ever-increasing difficulties. Severe hæmorrhage may be encountered from blood sinuses; the only way to stop this is by muscle stamps. A piece of temporal muscle is usually available, and may be squeezed into a thin stamp and placed on the torn sinus while the sucker displays the tear. If it is held gently in place with a gloved finger for a few minutes, adhesion of the stamp and control of the bleeding result.

Removal of Foreign Bodies.

The majority of foreign bodies encountered were bone fragments, which were often literally sprayed deeply into the brain from a tangential wound. Metallic foreign bodies were nearly all non-magnetic. The removal of most foreign bodies is a difficult matter, because the brain gives no resistance against which the body may be grasped, and clumsy handling will only drive it further away and may easily cause perforation of the ventricle—a major tragedy. Ordinary types of forceps tend to push a slipping object away as they close on it. Forceps with a sugar-tong action avoid this difficulty. Deep or inaccessible foreign bodies should on no account be sought after.

Localized Brain Damage as an Indication for Surgical Measures.

Misconceptions of cerebral pathology are responsible for unnecessary surgical procedures. There sometimes seemed to be an impression that a hemiplegia betokened a clot compression, and unnecessary exploratory operations were performed. It is well to remember that the outstanding feature of clot compression is loss of consciousness, paralysis being of secondary importance. It is more than improbable that a man who remains fully conscious is suffering from clot compression. Another explanation should be sought for the paralysis. These signs of localized brain damage were common after tangential wounds, which often had not even injured the skull.

A gunner reached the base hospital ten days after being wounded. He had received a head injury, and after a short period of unconsciousness he had recovered with a right hemiplegia. On admission to hospital, he had obvious weakness of the face, arm and leg. He had an open scalp wound of the right parietal region, with no bone injury or penetration. He progressed to full motor recovery in about six weeks, though residual loss of joint sense persisted.

Such brain damage was often extensive and recovered slowly.

A private soldier had a penetrating wound of the right parietal region, and a small metallic foreign body was lying to the left of the sagittal plane. There had been practically no unconsciousness, but he suffered immediate complete left hemiplegia and considerable disturbance of voluntary power in the right arm. He had been subjected to an unnecessary decompression operation. After some weeks his right side had completely recovered and power was returning to his left side.

If the surgeon remembers that increasing unconsciousness is the cardinal sign of compression, such patients will be spared the danger of unnecessary operations.

Compound Gunshot Wounds Involving the Orbital Roof.

When a perforating wound of the orbit has occurred, one must be aware that penetration of the cranial cavity through the orbital roof is a possibility. This at a later stage may result in meningitis or brain abscess. Four cases were treated in which this complication of severe damage to the eye was encountered. In two of the cases the eye had already been removed when brain tissue was noted in the orbital cavity. The procedure then adopted was to retract the lids well, gently wash and suck out the pulped brain, and remove loose fragments of the orbital roof. It is a good practice then to suture the lids together. In two cases in which this treatment was carried out a little cerebro-spinal fluid seeped out between the eyelids for a few days, and there was then no further anxiety.

One patient whose lids had not been sutured was seen much later at the base hospital, and had developed an intraorbital *fungus cerebri*. He had severe meningitis; the cerebro-spinal fluid pressure was over 300 millimetres and the cell content was 1,200 per cubic millimetre. With general treatment he recovered and the *fungus cerebri* subsided.

General Treatment of Wounds of the Head.

There is no doubt that the sulphonamides save many casualties with head wounds, and thus preserve for surgeons some problems of the late treatment not heretofore encountered. Sulphanilamide should be administered at the first dressing station. Confusion and difficulty were often caused by patients having no record of their early dosage. Special slips should be attached to the field medical card for this purpose. Where a patient's evacuation is a matter of doubt or chance, it is wise to give a very large initial dose of "M & B 693" or sulphonamide.

We found it necessary to make estimations of the concentration of sulphanilamide in the blood of patients arriving at the base with infective complications. A concentration of under 7·0 milligrammes *per centum* is too low, and the dosage should be "pushed" until a satisfactory concentration is obtained. At the same time, the leucocytes should be watched. Lumbar puncture is performed in all cases when signs of intracranial infection are present, and the pressure is measured by a manometer and reduced to normal level. This should be done every day or more frequently if the pressure is high. It both affords relief and aids in the recovery process. The fluid intake and nutrition of patients are often a difficulty, as they may be vomiting or non-cooperative. When this is the case glucose and saline solution should be given intravenously and soluble "M & B 693" may be administered in it.

Summary.

Experiences of head surgery in Western Desert warfare are thought to be applicable to certain Australian conditions.

The liability to infection is discussed, and the long period over which radical primary treatment can be applied is emphasized.

Every effort must be made to excise and close all head wounds in contradistinction to wounds in other areas.

Some technical points of primary treatment and immediate after-care are considered.

Reports of Cases.

FAMILIAL PERIODIC PARALYSIS.

By JOHN MCGEORGE, M.B., Ch.M., D.P.M.,
Sydney.

Clinical Record.

A.G., aged sixteen years, gave a history of "turns" since the age of ten years. These latterly have taken the form of complete paralysis of the limbs and trunk, coming on at irregular intervals and lasting a few days. The attack at the age of ten years was of a transient nature and consisted merely of slight muscular weakness. The first definite manifestation occurred at the age of fourteen years, when he was riding a bicycle and lost the use of both legs. Since then he has had three severe attacks with complete paralysis, and several minor ones in which only one limb or a muscle group was affected. He has noticed that sitting for any length of time in the one position is likely to precipitate attacks. He has been in the Royal Prince Alfred Hospital on three occasions, two of which were actually during the period of paralysis.

Physical Examination.

When examined between the attacks, the patient is seen to be an obese lad, five foot six and a half inches in height, and weighing 10 stone 13·5 pounds. His general condition is good and no muscular weakness is present. His reflexes are normal; his blood pressure is 130 millimetres of mercury, systolic, and 70, diastolic. His fundi are normal. The only abnormality of note is a fine tremor of the fingers.

During the attacks complete paralysis of the trunk and limbs is present, and his respiration is abdominal in type. He is unable to move his body or limbs or to turn over in bed; but movements of the head and eyes, swallowing and speech remain unimpaired. Muscle tone is retained, but his knee, ankle, biceps, triceps and jaw jerks are all absent. The abdominal reflexes are also absent. Skin sensibility is unchanged; sensations of touch, pain and passive position are unaltered. The function of the sphincters is normal.

Special Investigation.

A blood count revealed that the red blood corpuscles numbered 5,310,000 per cubic millimetre and the hæmoglobin value was 15·5 grammes *per centum*, which is 107% of the normal; the colour index was 1·0. The white blood corpuscles numbered 5,900 per cubic millimetre and a differential count showed that 50% were neutrophile, 33% were lymphocytes, 6% were mononuclear cells, 8% were eosinophile cells and 3% were basophile cells. The blood sedimentation rate was 2·5 millimetres, which is normal. The basal metabolic rate was −3%. X-ray examination of the skull revealed that the *sella turcica* was normal in size and shape, but the frontal sinuses were unusually large (a condition seen in acromegaly), and prognathism seemed to be present. An X-ray examination of the chest revealed no persistent thymus. Neither the Wassermann test nor the Kline precipitation test produced a reaction. During the attack, all the muscles of the upper and lower limbs gave normal responses to faradism.

Family History.

The patient's father died at the age of thirty-five years, having suffered from a similar condition from the age of eighteen years. A paternal aunt, still living, is also affected; but the onset in her case was at the age of forty years. One of her sons died from pneumonia (possibly inhalational) during a period of paralysis, and another son is a victim of the same condition. In both cases the first symptoms developed at about the age of eighteen years. Another son and two daughters are quite healthy. The patient has one brother who is perfectly normal. It has not been possible to trace any other cases occurring in either branch of the family.

Comment.

There can be little doubt that this is a case of familial periodic paralysis. The family history, the periodicity of the attacks, their relatively brief duration and the complete recovery in the intervals, are all strongly in favour of this diagnosis. The fact that power is completely regained excludes the possibility that one of the common muscular dystrophies or a lesion of the spinal cord is responsible.

This disease is a comparatively rare one, and was first described by Cavare in 1863. Sporadic cases may occur; but it has been estimated that 81% of cases have a familial incidence. The condition is transmitted by both sexes, but males are affected twice as frequently as females. The ætiology is obscure, and many theories have been advanced to account for it. It has been regarded as nervous in origin or due to some local change in the muscles, such as alterations in the chemical or physical constitution of the muscle plasma. A relationship to migraine has been postulated without any strong evidence to support it. Other theories include ischæmia of the muscles, disorders of metabolism, interference with lymphatic circulation and muscular dystrophic changes. Recent research tends to indicate that there is a fall in the serum potassium level during an attack and that this interferes with the passage of the nerve impulse and also with the response of the muscle fibre.

It is interesting to note that in this case the electrical reactions were normal. Such is not usually the case. In other respects the findings were consistent with other reported cases.

Good results have been claimed in the aborting of attacks by the oral administration of 12 grammes of potassium chloride, the paralysis clearing up in an hour.

The prognosis is not unfavourable; the attacks attain their maximum severity between the ages of twenty and thirty years, after which they tend to lessen and ultimately to disappear.

Reviews.

SELF-ANALYSIS.

THE fact that Freud wrote a foreword to the book "A Practical Method of Self-Analysis", by E. Pickworth Farrow, M.A., D.Sc., is sufficient guarantee that it is worth reading.[1] He wrote: "The author of this book is known to me as a man of strong and independent intelligence who, probably on account of a certain wilfulness of character, could not get on well with the two analysts with whom he experimented. He then had recourse to a consistent application of the process of self-analysis which I had once used myself in order to analyze my own dreams. His results deserve notice, especially because of his special individuality and his technique."

The author underwent two short courses of psycho-analysis totalling two hundred hours, and then determined to be his own analyst: instead of saying whatever thoughts came to his mind in the presence of a trained psycho-analyst, he adopted the plan of writing them down and interpreting them himself. Subsequently, he substituted talking to himself for such written "free-associations". He claims that after following this technique he reexperienced several emotional disturbances of his early childhood and that his health improved as a result.

Freud might have stated in his foreword that practising analysts carry out a certain amount of self-analysis subsequent to the psycho-analysis which constitutes the main part of their training, and which may last nearly one thousand hours. Though, then, the author failed to obtain the benefit of analysis on ordinary lines, he has made a definite contribution to technique by carrying out self-analysis more systematically. If, however, he intends to recommend the general adoption of self-analysis without a preliminary ordinary course he is ignoring the fact that, though a genius may make progress by self-analysis alone, Freud's colleagues thought it worth while to be analysed by others for a considerable time. On the other hand practising analysts might well adopt a more systematic form of self-analysis, such as that developed by the author. Self-analysis might also be used in future as an adjuvant to ordinary analysis for patients who exhibit excessive fear of the temporary dependence entailed by the treatment. The author, however, demonstrates by his remarks that he did not undergo sufficient ordinary analysis.

[1] "A Practical Method of Self-Analysis, Enabling Anyone to Become Deeply Psycho-Analyzed Without a Personal Analyst", by E. Pickworth Farrow, M.A., D.Sc., with foreword by the late Professor Sigmund Freud, M.D., LL.D.; 1942. London: George Allen and Unwin Limited. Crown 8vo, pp. 170. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 5, 1942.

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SULPHONAMIDES, THE MEDICAL PRACTITIONER AND THE WAR.

No one will deny that there are fashions in medicine, particularly in the prescribing of drugs. It is not always the same type of person who sets the fashion. Sometimes a prominent practitioner who has opportunities for observation of the results of therapy in a large number of cases reads a paper before a medical gathering or has an article published in a journal setting out his results with, let us suppose, a restrained and reasonable appraisal of the value of the medicament used. He may, of course, allow his enthusiasm to get the better of his judgement and make unjustified claims for his method of treatment. But it will not really matter much in the long run whether he is cautious or not, for those who hear his words or read them in cold print, are as a rule easily impressed and the remedy with which the prominent practitioner has had success must be "tried out". This trial is often followed by a satisfactory outcome which is attributed to the remedy—the *post hoc* becomes almost automatically the *propter hoc*. The fashion may be set by a manufacturing firm which grips the imagination either by a subtle form of advertisement or by the choice of a name with a mellifluous sound. The following of a fashion in therapy need not necessarily be detrimental to the welfare of the patient—the *vis medicatrix naturæ* often takes care of that—but everyone will agree that fashions in any walk of life dull the critical faculties of those who follow them. The fashion-following therapeutist therefore does well to take stock from time to time both of his knowledge and his understanding.

During war an additional factor enters into this question and this factor is the availability of the medicament to be used. This is particularly true of Australia where many important products have to be imported. The Medical Equipment Control Committee was set up in order to deal with the supply of therapeutic agents and to ensure as far as possible that the vital needs of the medical profession and of the community would be met. The success of its endeavours is reflected in the relatively slight inconvenience that has been experienced by practising members of the medical profssion. For this reason communications and requests from the committee should receive the undivided attention of every medical man and women. We have recently declared that the committee makes no request to the profession without just reason and it must be clear to everyone that categorical reasons for requests cannot always be given.

The Medical Equipment Control Committee, as a result of a request from one of the Australian Branches of the British Medical Association, has had occasion to make a statement regarding the supply of certain drugs of the sulphonamide type. The facts must be brought to the notice of practitioners. Apparently certain doctors have had difficulty in obtaining supplies of sulphathiazole. Supplies of this drug have been short, though we understand that the firm which holds the proprietary rights for its importation and sale has recently landed one million tablets. That is not the point. The point is that medical practitioners are switching over from sulphapyridine ("M & B 693") often without any reason other than the fact that other people are doing the same. Large stocks of sulphapyridine are held in Australia and a million tablets of sulphathiazole will not last long if a "fashionable" run is made on them. It has been stated that sulphathiazole is more readily obtainable in America than sulphapyridine and that this is one of the reasons why it is used more often in that country than the latter drug. Another suggestion is that Australian practitioners are switching over to sulphathiazole as a result of conversations with American physicians now in the Commonwealth. Whether these suggestions are correct does not really matter. What has to be considered is the value of one drug as compared with that of the other. References to this subject will be found in the pages of every medical journal dealing with therapeutics and medicine. In the "Abstracts from Medical Literature" published in this journal during the last year or two repeated mention is made of it. In general terms it may be stated that vomiting is more frequent with sulphapyridine than with sulphathiazole. At the same time the vomiting is seldom sufficiently severe to prevent the administration of adequate doses by mouth or to interfere with fluid intake. Again, though difficulties may occur with either drug, skin rashes, fever and renal complications are possibly commoner with sulphathiazole. These renal complications may be serious and a number of deaths have been reported. Serious blood disorders, such as agranulocytosis, may be produced by either drug and no means of preventing them has been discovered. Though some observers hold that sulphathiazole is the more potent of the two drugs, it will be admitted by many practitioners that for most practical purposes one is as effective as the other. Those who agree with this view will, because of the present position regarding supplies, use sulphapyridine in preference to sulphathiazole. Those who hold that there are circumstances in which use of sulphathiazole is indicated—and we believe that both they and the circumstances are few—will be anxious to conserve stocks. They will, like the group already mentioned, use sulphapyridine on every possible occasion in preference to sulphathiazole. They will recognize that occasional vomiting will not matter; they will rightly insist that it is better for one patient to

vomit than for another to be deprived of a drug which in certain special circumstances is the one which should be used.

Two other drugs of the sulphonamide group are being brought into use and are being mentioned in many journals; these are sulphaguanidine and sulphadiazine. Of these two, the former is being used in intestinal infections (see THE MEDICAL JOURNAL OF AUSTRALIA, March 21, 1942, page 351); in certain respects it is still in the experimental stage. Sulphadiazine is at present being used freely in America. It is stated to have a low toxicity and to possess certain therapeutic advantages; clinicians, however, should realize that the information available is not really sufficient to allow its ultimate place in chemotherapy to be determined. The point about these two, sulphaguanidine and sulphadiazine, is that their popularity will almost certainly increase—they will become fashionable—and, rightly or wrongly, the present vogue for sulphathiazole will abate. The lesson should be fairly obvious. The supplies of drugs in this country cannot be arranged during wartime to meet the dictates of fashion. The pseudoscientists who mark their "progress" by believing everything that they read about a new drug and most of what they hear, will resent suggestions that they are following a fashion. They must be honest with themselves if they can and try to cultivate in therapeutics the common sense which is so necessary during a war and in a country situated as Australia is.

REST FOR DOCTORS.

EVER since the outbreak of war reference has been made from time to time in these pages to industrial fatigue and an effort has been made to show that continuous work in long shifts and without proper rest pauses leads in the long run to impaired efficiency, that is to say, to bad work and ill health of the worker. We have also tried to show that those who labour with their hands in factories and other industrial undertakings are not the only folk who can be regarded as workers. Not a great deal of argument is required to establish the thesis that workers of one type are just as likely to suffer fatigue as workers of another and that the quality of their work is just as likely to deteriorate when they are fatigued. At the present time doctors, particularly those in general practice, are working harder than they have ever worked before. A large percentage have joined the ranks of the armed forces and those left behind are trying to cope with the resulting increase in private and hospital practice. Not only this, but recruits have had to be examined, personnel of the National Emergency Services have had to be trained and lectures given to them, and assistance has been called for in other branches of the war effort. In times of peace the life of a medical practitioner is arduous, not only on account of the heavy responsibilities that are carried, but also because of irregular hours and irregular meal times and because of the strain of being always on duty. Doctors, then, are just as likely as anyone else to suffer from fatigue; their efficiency is certain to deteriorate unless some provision for rest and relaxation is made.

This subject has caused some concern to the Council of the New South Wales Branch of the British Medical Association and Dr. R. J. Verco, President of the South Australian Branch, made reference to it in his recent President's address. It is one which medical practitioners have to consider and about which they will have to take some action. Two things must be remembered; one is that the efficiency of the medical service to the public must be maintained, and the other that a twenty-four-hour-a-day service must be available for the treatment of patients suffering from urgent conditions. It is foolish for practitioners to think that they can carry on indefinitely without a rest. They would not allow their patients to attempt anything so foolish. They have to remember that if some plan can be evolved for the provision of time off duty, they are doing a disservice to their patients not to adopt it. To leave it to each practitioner to take a rest when he or she can get it is a haphazard arrangement and most unsatisfactory. Whatever is done must be done on a collective or organized basis. The New South Wales Council thinks that the organization should be undertaken in that State by the local medical associations. The idea is a good one and in other States local associations or subdivisions of the Branch might be the organizing body. The local body of practitioners in these circumstances would draw up a roster of week-end duties in such a way that week-end leave would come round regularly for each practitioner in the area. In view of the shortage of *locum tenentes* the principle might be extended to evening consultation hours, and, when local conditions were suitable, to annual leave. Practitioners in one-man towns could not well share in such an arrangement. Some arrangement should be possible by which a practitioner who was available for *locum tenens* duty could be reserved to relieve in turn the practitioners in a group of one-man towns. This might mean collaboration between two or more local groups or associations of doctors. To secure such an arrangement for one-man towns would be difficult; it is unlikely that any central body or any group of bodies would entertain the idea until practitioners in larger centres had on their own initiative organized or tried to organize a regular roster of leave. Thus the matter is in the hands of the profession itself and the need for action is urgent in the interests of the patients and of the health and efficiency of doctors.

Current Comment.

DRUGS AFFECTING THE MOTILITY OF THE COLON.

PARALYTIC or adynamic ileus, because of its seriousness and its resistance to treatment when fully established, is, as is well known, one of the bugbears of surgery. The frequency of this post-operative complication has directed attention to it, and many attempts have been made to prevent its occurrence or to treat it, but only with varying degrees of success. These efforts include the experimental work of laboratory workers, prophylactic measures undertaken before, during and after operation, and therapeutic and operative procedures once its presence is suspected or discovered.

It is almost impossible to keep track of the numerous papers on adynamic ileus and its treatment which appear in the literature, but that of H. F. Adler, A. J. Atkinson and A. C. Ivy[1] is worthy of notice. These authors have reviewed the literature on drugs used to stimulate motility

[1] *Surgery, Gynecology and Obstetrics*, April, 1942.

of the human colon and have concluded that most reports favour the use of prostigmine, but that although one favourable report of the use of ergotamine has been published, this drug has been neglected. These authors carried out experiments on trained dogs and ambulatory male adults with colostomies; they employed the balloon technique and investigated the effects of various drugs on the motility of the colon. They point out that it is important to realize that two types of motility occur in the colon, propulsive and non-propulsive, and it is necessary to differentiate between them. It has often been shown that morphine sulphate increases the total motility and tonus of the distal part of the colon and ileum of man; but this increase in motility is of the non-propulsive type, whereas the propulsive motility and discharge of the contents of the colon are considerably decreased by this drug. Again, although pituitrin causes little or no increase in the non-propulsive motility, it gives rise to a definite increase in propulsive motility. On the other hand, both types of motility are augmented by prostigmine. As Adler, Atkinson and Ivy point out, the chief criticism levelled at the drugs used to stimulate motility of the human colon is their relative ineffectiveness in doses which would not be accompanied by side-actions such as nausea, vomiting, salivation, headache and cardio-vascular changes. By combining these drugs Adler and his co-workers attempted to obtain supplementary and synergic actions so that small doses might be employed and these side-effects avoided. The combination of physostigmine sulphate with pituitrin resulted in the addition of the effects on the bowel of each, apparently partly owing to the prompt transient action of pituitrin followed by the more delayed and prolonged action of the physostigmine. The same was true of combinations of prostigmine methyl salicylate with pituitrin or with ergotamine tartrate. According to these authors the most effective combination from the point of view of duration of effect was ergotamine tartrate 0·25 milligramme, prostigmine methyl salicylate 0·25 milligramme, and surgical pituitrin 1·25 units. The effect of this mixture extends over six to eight hours and it is suggested that it might be of use for the treatment of adynamic ileus or of megacolon. These authors also concluded that of any one single drug used to stimulate the colon in post-operative distension or adynamic ileus prostigmine is easily the best.

In the paper under review Adler and his co-workers have confined themselves to an investigation of drugs which stimulate the bowel. Such drugs have a definite place in the treatment of post-operative distension and ileus; but until the ideal drug which restores normal intestinal movement in the absence of any effects is discovered, the method of permitting a period for rest and recovery of the tired bowel and the tired patient is more often to be preferred.

THE BIOLOGY OF EPIDEMICS.

THE Croonian lecture delivered by W. W. C. Topley, F.R.S., in July, 1941, on the biology of epidemics has only recently been published in the *Proceedings of the Royal Society*, England.[1] This lecture gives a concise and admirable summary of the author's long-continued researches on the laws governing epidemics as revealed chiefly in experiments on mice. The number of animals used and the ease of diagnosis allowed satisfactory inductions to be formulated. Three diseases to which mice are naturally prone were employed in the inoculations, namely, pasteurellosis, a bacterial disease of the respiratory tract, mouse typhoid and a virus disease, ectromelia. The experiments included and paid attention to varying the rate of addition of susceptible mice, changing from continuous to discontinuous contact, group to group infection, natural and artificial immunization, the epidemic potency of different strains of the same parasite, genetic differences in resistance in the same host and the effects of dispersal on an infected herd. In the last mentioned the interesting result was obtained that if the epidemic has just started, then dispersal into large groups has no effect, but dispersal into small groups reduces the morbidity; if, on the other hand, the epidemic is well under weigh, dispersal makes no difference. Under the head of varying potency it was shown that one strain of a bacterial parasite may kill more hosts than it immunizes, whereas another can immunize more than it kills. In protracted epidemics the author is of the opinion that variation in epidemicity or virulence plays little part in determining fluctuations of morbidity. An interesting inquiry into infections, especially naso-pharyngeal, in boarding schools, both boys' and girls', showed convincingly that in every term in every year there is a primary peak incidence between the second and fourth week of term, usually between the third and fourth. This cannot be seasonal in origin as the same peak is displayed in each term and is obviously due to the reaggregation of boys and girls after the dispersal of the holidays with the consequent effective contacts. "Epidemics of this kind are at the moment part of the price we must pay for education."

The most arresting conclusion of this careful inquiry is that the biological system of an epidemic is in unstable equilibrium, shifting now to the advantage of the parasite, now to that of the host. It is unnecessary to aim at the reduction of the probability of infection to zero; if a pronounced diminution of effective contact is achieved then the balance is tipped over and the disease will decline to negligible proportions or may even disappear. The author is fully aware that there are complexities which he has not examined, as for instance when insect vectors are involved; the probability of infection and spread of malaria has been mathematically handled by other authors. Curiously enough he omits mention of bacteriophage as an agent in the subsidence of epidemic virulence.

[1] *Proceedings of the Royal Society*, B, Volume CXXX, May, 1942, page 337.

THE USE OF PHOLEDRINE OR "VERITOL" AT MAJOR OPERATIONS.

IN September, 1939, attention was drawn in these pages to a paper in *The British Journal of Surgery* of July, 1939, by Harold Dodd and Gerald Merton on the use of "Veritol" as a blood pressure stimulant for use during and after operation. Dodd and Merton were introduced to "Veritol" by a statement made by Sir Stanton Hicks in *The Australian and New Zealand Journal of Surgery* of October, 1937, that "Veritol" was the nearest approach that had been obtained to the ideal physiological restorative of the circulating blood volume, provided the depots were not depleted. Dodd and Merton found that the drug was a reliable and satisfactory restorative of blood pressure, even when given intramuscularly; its only weakness was that its effect did not last long enough. In *The Lancet* of February 24, 1940, Dodd reported investigations into several drugs used in surgery to raise blood pressure and reported that "Veritol" was the most satisfactory. In *The Lancet* of March 23, 1940, G. Schoenewald, A. Schweitzer and C. C. Steel reported experimental observations with "Veritol" on cats, which, if applied to man, were not in favour of its clinical use. Harold Dodd bases a recent article[1] on clinical findings during and after major operations as shown in nearly 300 charts, depicting the use of the drug, which he calls by the non-proprietary name of pholedrine. This paper should be studied by surgeons. Shortly, Dodd analysed the blood pressure charts of 89 patients who received the drug during major operations. The effective dose was 0·2 to 0·25 cubic centimetre intravenously, 1·0 cubic centimetre intramuscularly, or 0·2 to 0·25 cubic centimetre intravenously *plus* 0·75 to 0·80 cubic centimetre intramuscularly. The amounts in a single dose raised the pressure to within 25% of the normal. Repeated doses, up to five, were effective. Apart from minor variations in the pulse, there were no ill-effects. Apparently some experience in assessment of dosage and estimation of the patient's condition is necessary.

[1] *The Lancet*, April 25, 1942.

Abstracts from Medical Literature.

PHYSIOLOGY.

Vital Capacity Studies in the Aged.

ISIDORE MILLER (*The Journal of Laboratory and Clinical Medicine*, March, 1942) describes the result of vital capacity determinations made on 744 men, forty to ninety-four years of age. There was a decrease in vital capacity from 3,150 cubic centimetres (79% of normal) at forty to forty-four years to 2,400 cubic centimetres (62%) at ninety to ninety-four years. The vital capacity for the group as a whole was 2,780 cubic centimetres (71%). The causes of the decreased vital capacity are loss of elasticity of the pulmonary tissue, the lessened mobility of the thoracic cage and diminished muscular power. Pulmonary fibrosis, pulmonary tuberculosis, pulmonary emphysema and bronchiectasis gave vital capacity readings below normal for age groups. Persons with compensated heart conditions gave normal vital capacity readings. Lowered figures were found in men with systolic blood pressure of 200 millimetres and over. The author states that although a normal vital capacity does not denote the absence of pulmonary pathological change, a low vital capacity is significant.

The Origin and Nature of the Cabot Ring Bodies of Erythrocytes.

E. M. SCHLEICHER (*The Journal of Laboratory and Clinical Medicine*, May, 1942) discusses the controversy concerning the origin and nature of Cabot ring bodies, pointing out that Cabot's hypothesis of the ring bodies representing "nuclear remnants" was diametrically opposed to the tinctorial behaviour of the structures; he then proceeds to detail his own experiments. Many more Cabot ring bodies were to be seen in blood films which were allowed to dry by slanting than in control films which were dried in the standard manner, care being taken that the angles of slant were not great enough to encourage concentration of red blood cells, but only plasma. The author found that with one particular specimen of blood from a patient suffering from generalized Hodgkin's disease, the number of ring bodies could be varied at will, but this could not be accomplished to the same extent in all blood samples taken from patients with severe anæmia. Further experiments were designed to determine the reason for the fluctuation of dimensions, configurations and affinity to specific cytoplasmic dyes of the ring bodies; these showed that some of the ring bodies were at or near the surface, and seemingly part of the envelope, whereas others seemed to belong to a deeper layer. Some of them were superimposed on others, giving a wide variation in size and shape, whilst those ring bodies which stained irregularly were shown to be composed of irregular thick bars or tiny globules. From this the author concludes that the staining variability depends upon the character and quantity of the substance that makes up a ring, the molecular thickness of such a structure, and the momentary physical state of ring constituents. This conclusion, he states, was proved by further experiments in which egg white was used instead of blood films, and he also shows that the ring bodies are protein derivatives of the envelope and cytoplasm. In these experiments with egg white, the author was able to produce Cabot ring bodies, which were "a replica in every respect" of those found in blood films. Using egg yolk tinted with Wright's stain, he was able to produce "the phenomenon known as diffuse and punctate basophilia", and he expresses the opinion that the various tinctorial shades of diffuse basophilia are apparently the expression of a more or less homogeneous state of protein and lipoids, and that punctate basophilia may be interpreted as the result of disassociation of these substances, or the independent reaction of the laminated lipoid component. Thus it is understandable why a ring body can appear in an erythrocyte exhibiting either diffuse or punctate basophilia. The use of special methods with normal mature erythrocytes gave simulacra of both ring bodies and punctate basophilia at will, and the author gives an account of the seeming formation of Cabot ring bodies, concluding that they are neither nuclear remnants nor identical with the nuclear membrane, but are laboratory creations, the expression of cellular degeneration induced by hæmolytic agents.

The Spleen and Blood Regeneration.

W. O. CRUZ AND F. S. ROBSCHEIT-ROBBINS (*The American Journal of the Medical Sciences*, January, 1942) have examined the blood of three dogs which had been subjected to splenectomy and nineteen dogs in which this operation was not performed. In dogs with no spleen which had been made anæmic by bleeding or by acetylphenylhydrazine injections, the number of normoblasts during the first phase of the regenerative period was four times greater than in the dogs which had not undergone splenectomy. Sometimes the number was even higher. The number of primitive erythroblasts (the basophilic erythroblasts of Ferrata) was much higher in the animal with no spleen; in some cases the number reached 40% of the nucleated red cells in the circulation. The authors think that their findings suggest a relationship between a function of the spleen and the maturation of the erythroblast in the bone marrow on a factor acting upon the launching of new red cells into the general circulation.

Boyle's Law, Pneumothorax and Aviation.

W. R. LOVELACE AND H. C. HINSHAW (*The Journal of the American Medical Association*, April 11, 1942) have discussed the effect of decreased barometric pressure (high altitude) and of increased barometric pressure in a paper dealing with the dangers to which persons with pneumothorax are exposed when they travel by air. They have demonstrated this effect by radiological studies. They point out that in accordance with Boyle's law free gas within the human body tends to increase in volume when a person ascends in altitude and to decrease in volume when the atmospheric pressure is increased. Although complaints have been few when patients with pneumothorax have travelled by aeroplane, their medical attendants would be alarmed if an artificial pneumothorax was refilled with a litre of air or more, yet this is precisely what should happen theoretically when a patient with a large pneumothorax cavity ascends to the moderate altitudes frequently attained by commercial aeroplanes. The authors have experimented with a low pressure chamber capable of accommodating the patient and the X-ray apparatus, and in this way have been able to simulate any desired altitude. They found a considerable variation among different patients. When the pneumothorax did not expand according to Boyle's law the failure was explained by thickened pleura, adhesions, small capacity or other mechanical factors. Large changes in volume appeared to be very hazardous, but were not always productive of symptoms. When symptoms did appear they were attributed to traction on adhesions, or to reduction in vital capacity as a result of excessive collapse.

The Purification of Thrombin.

H. MILSTONE (*The Journal of General Physiology*, May 20, 1942) writes that the coagulation of blood is customarily considered in two stages. During the first stage a series of reactions results in the activation of prothrombin to thrombin. In the second stage thrombin brings about the conversion of fibrinogen to fibrin. The mechanism of these reactions has yet to be unravelled. As a part of this investigation the author has studied the purification of thrombin. He shows that under certain conditions crude prothrombin changes to thrombin without the addition of extraneous activators and in the absence of ionic calcium. Thrombin is soluble in a 0·45% solution of ammonium sulphate, whereas crude prothrombin is not. Partially purified thrombin is comparatively stable in concentrated ammonium sulphate solutions at pH 5·2. The method described by the author is based on these facts and, he states, yields a thrombin preparation the specific activity of which is 100 to 175 times the potential specific activity of whole plasma.

BIOLOGICAL CHEMISTRY.

Proteolytic Digestion in the Spleen.

SAMUEL P. HICKS AND EUGENE L. OPIE (*The American Journal of Pathology*, March, 1942) have shown that normal splenic tissue contains an enzyme capable of splitting protein in the presence of a faintly acid medium. The activity of this enzyme could be measured only after the blood had been removed from the spleen by perfusion. Parallel with the increase of phagocytosis of washed red and white corpuscles following introduction of an excess of these cells into the circulating blood, there was increased proteolytic activity of splenic tissue, measured by its ability to digest casein. The increased proteolytic activity reached a high level soon after injection of blood corpuscles, maintained this elevated level for periods of from six to twenty-four hours after injection of corpuscles and had returned to normal forty-eight hours after injection. The enzyme showed maximum activity at pH 6·7 and was almost completely inhibited by an alkaline reaction of pH 7·4. In these characters it resembled the proteolytic enzyme present in the mononuclear

phagocytes of inflammatory exudate. Parenteral digestion was considered a significant function of the spleen and proceeded with increased activity in some forms of "acute splenic tumour".

Serologically Active Phospholipid from Beef Heart.

MARY C. PANGBORN (*The Journal of Biological Chemistry*, March, 1942) has isolated and purified a new phospholipid from beef heart. The name cardiolipin is suggested for the substance which is essential for the reactivity of beef heart antigens in the serological test for syphilis. A properly balanced mixture of cardiolipin with lecithin and cholesterol exhibits a specific complement-fixing activity with serum from syphilitic patients and this is closely similar to the activity of beef heart extracts prepared for routine diagnostic use. None of the three substances in the mixture was found specifically active in the absence of the other two. No substance similar to cardiolipin has previously been isolated from animal tissue. Cardiolipin apparently represents a class of phospholipids not previously obtained in a pure state, compounds consisting of a polysaccharide phosphoric acid esterified with fatty acids.

Relation of Aspartic Acid and Glucosamine to Growth.

WILLIAM C. ROSE AND SCHEURING S. FIERKE (*The Journal of Biological Chemistry*, March, 1942) have shown, by the use of diets devoid of proteins, but containing mixtures of highly purified amino acids, that aspartic acid and glucosamine are dispersable components of the food. Rats deprived of either of these compounds increased in body weight just as rapidly as did litter mates receiving a similar ration supplemented with the compound in question. Seventeen components of proteins have been definitely classified with respect to their significance for growth. Of these, phenylalanine, theonine, leucine, isoleucine, methionine, valine, lysine, troptophane and histidine are indispensable. On the other hand, tyrosine, norleucine, glycine, serine, cystine hydroxyglutamic acid, citrulline and alanine are dispersable components, since they can be synthesized *in vivo* out of materials ordinarily available.

The Absorption of Fat.

RAYMOND REISER (*The Journal of Biological Chemistry*, March, 1942) has studied the absorption of fat by the mucosa of the duodenum, using the pig for his experiments. There was no change in the phospholipid or cholesterol content of the mucosa during absorption. In the fasting animal there were no triglycerides in the duodenal mucosa and only small amounts, if any, during absorption. During fasting, the mucosa contained about 2·5% of free fatty acids (calculated on the dried weight) and this amount was approximately doubled after ingestion of fats. This change in fat content would by no means account for the very great increase in stainable fat observed in histological studies. This discrepancy is explained by the view that in the resting mucosa the lipids are a definite part of the protoplasm and are not stainable, but that during absorption there is a change in their physiochemical state and they then exist as individual compounds. In that state they are stainable, and hence on histological examination there appears to be an enormous increase in fat as compared with the fasting state.

Dietary Hepatic Injury in Rats.

PAUL GYÖRGY AND HARRY GOLDBLATT (*The Journal of Experimental Medicine*, April, 1942) have shown that experimental dietary hepatic injury (diffuse or focal necrosis and cirrhosis in rats, with or without ascites and pleural and pericardial effusion) is determined by the dietary factors instrumental also in the production of fatty infiltration of the liver, and thus opposed to the lipotropic activity of casein. Thus, rats maintained on a diet poor in casein with a moderately high or high content of fat and without choline regularly exhibited hepatic injury after between 100 and 150 days. Supplements of l-cystine had an aggravating effect on the production of cirrhosis of the liver, whereas a supplement of choline alone reduced the severity and incidence of hepatic injury. The combined administration of l-cystine *plus* choline or of de-methionine proved highly effective in preventing injury to the liver. Rats with dietary hepatic injury exhibit, in sequence, changes that vary from diffuse necrosis, resembling human acute or subacute yellow atrophy, to advanced portal cirrhosis. The authors point out, by analogy, the unquestionable similarity of the ætiological conditions that prevail in the most important form of human cirrhosis, namely, that observed in alcoholics. Low intake of protein combined with insufficient supply of the vitamin *B* complex (including choline) is a prominent feature of the daily diet of persons addicted to alcohol and compares with the leading conditions of dietary cirrhosis in rats. Thus the assumption of a specific injurious effect of alcohol here becomes superfluous, just as it does in pellagra or the beriberi of alcoholics.

Pantothenic Acid Deficiency in the Dog.

A. E. SCHAEFFER, J. M. MCKIBBIN AND C. A. ELVEHJEM (*The Journal of Biological Chemistry*, April, 1942) have shown experimentally that pantothenic acid is an essential requirement in the nutrition of the dog. The quantitative requirement for young growing puppies was found to be approximately 100γ of calcium pantothenate per kilogram of body weight per day. Pantothenic acid deficiency was characterized by sudden prostration or coma, usually rapid respiratory and heart rates, convulsions and gastro-intestinal symptoms. Post-mortem examination revealed fatty livers, mottling of the thymus, evidence of hæmorrhagic degeneration in the kidney and frequently gastritis or enteritis and intussusception. In some deficient dogs in which no intussusception was found at necropsy, vomiting attacks were so severe that fæcal material appeared in the vomitus. The possibility is suggested by the authors that intussusception in human infants may have a nutritional background. Preliminary studies of the blood chemistry showed that in deficient dogs, the blood glucose and chlorides were lowered and non-protein-nitrogen values were raised. These values returned to normal after recovery from the deficiency.

MEDICINE.

Toxic Effects of "Mapharsen".

EDWARD A. LEVIN AND FRANCES KEDDIE (*The Journal of the American Medical Association*, January 31, 1942) present a summary of the literature on the toxic effects of "Mapharsen". Only six deaths have been reported, two due to kidney damage, one due to hæmorrhagic encephalitis, two to aplastic anæmia, and one to acute agranulocytosis. There have been no deaths after approximately 35,000 injections at the University of California syphilis clinic between 1934 and 1940. Rapid injection has decreased the incidence of pain in the arm, which was a frequent complication when the drug was given slowly. Cutaneous reactions occurred in 13,769 patients who received 240,811 injections of "Mapharsen". Pruritus occurred in 84 cases, a ratio of 1:163, scaling and erythematous eruptions of moderate and mild degree occurred in 70, urticaria in 27, œdema of face and body in 14, exacerbation of previously existing dermatitis in five, exfoliative dermatitis in 16 cases. The incidence of mild gastro-intestinal reactions from "Mapharsen" was 1 in 15, of severe reactions 1 in 334. One in every 450 patients had jaundice during therapy. The jaundice was mild and disappeared within three to eight weeks. "Mapharsen" is less toxic than neoarsphenamine when administered in the usual manner. It is also less toxic when given by the continuous intravenous drip method. About 85% of patients who have mild or moderate cutaneous reactions to the arsphenamines can tolerate "Mapharsen". About 90% of the patients who have severe gastro-intestinal reactions to the arsphenamines can tolerate "Mapharsen" in therapeutic doses. No case of true nitritoid reaction from "Mapharsen" has been described.

Inversion of the "T" Wave and the Position of the Heart.

PAUL D. WHITE, FRANCIS L. CHAMBERLAIN AND ASHTON GRAYBIEL (*British Heart Journal*, October, 1941) discuss inversion of the *T* waves in lead II caused by a variation in position of the heart. They state that inversion of the *T* waves in lead II of the electrocardiogram, although most commonly the result of heart disease or toxic states, may be a normal physiological variation in occasional persons, particularly those of asthenic habitus with vertical hearts and prone to neurocirculatory asthenia. The most important factor in producing this *T* wave inversion is the position of the heart. It is found among patients in the sitting or standing position, and is corrected by recumbency or by elevating the diaphragm as at full expiration. Autonomic nervous influences comprise another factor, the low or inverted *T* waves then being attended by tachycardia; any cause of such stimulation, for example, excitement, can be responsible. Fear and anxiety may act through the production of overventilation with resultant alkalosis. Both heart position and nervous influences may be active in the same case. The relatively common occurrence normally of inversion of the *T* waves in lead II makes it imperative to recognize its existence in order to avoid erroneous diagnoses of heart disease.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 25, 1942, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, Dr. W. F. SIMMONS, the President, in the chair.

Anæsthesia and Shock.

DR. A. DISTIN MORGAN read a paper entitled "Shock and Anæsthesia in Relation Thereto" (see page 193).

LIEUTENANT-COLONEL A. C. THOMAS expressed gratitude to Dr. Morgan for his excellent, practical paper; the paper was so practical that there was very little left for him to add. However, Dr. Thomas wished to express his agreement with the contention that there should be more cooperation between the surgeon and the anæsthetist. He thought that it was always necessary for the anæsthetist to see the patient before operation. The anæsthetist certainly saw the patient during the operation, but he should also visit him after the operation, because it was only by doing so that he would be able in the future to assess what type of anæsthesia would be best in similar circumstances. There should be the fullest cooperation between surgeon and anæsthetist, both before and after operation. The subject of anæsthesia and shock was important at the present time, not only for war surgery, but also for civilian surgery. Perhaps it would be necessary for surgeons to see and deal with all degrees of shock. Colonel Thomas had recently been impressed with the differences in the treatment of shock as carried out when he was a resident medical officer and the treatment adopted at the present time. In his time a patient suffering from shock was put back to bed and the foot of the bed was raised; strychnine and pituitrin were used. Later they began to give saline solution *per rectum*, and towards the end of his career as a resident medical officer they were giving saline and gum solutions intravenously. Colonel Thomas went on to say that Dr. Morgan had well discussed the indications for the different kinds of fluid to be given intravenously (saline solution, plasma, serum). In reading the literature on shock, Colonel Thomas had always felt great admiration for the work of the commission of the British Ministry of Health, which, during the last war, had carried out investigations under shell fire and in No Man's Land on soldiers suffering from shock and hæmorrhage. It was necessary to distinguish between primary and secondary shock. Primary shock (neurogenic shock) occurred immediately after the trauma had been inflicted; it was a reflex vascular effect due to pain and psychic trauma. The purpose of the meeting was to discuss secondary shock, which came on generally from up to four to twenty-four hours after the infliction of the trauma. After having read Dr. Morgan's paper, Colonel Thomas thought it might be good to discuss briefly some of the physiological aspects of shock from the surgeon's point of view. Dr. Morgan had mentioned certain physiological features. Colonel Thomas did not intend to try to discuss the causes of shock; the subject was full of confusion and contradiction. For example, Dr. Morgan had spoken of the use of "Eschatin", or adrenal cortical extract. Some experiments had shown that adrenal cortex was necessary to combat shock; others had shown that it was not necessary. Some workers had shown that shock was due to adrenal hypofunction, others had shown that it was due to adrenal hyperfunction; others again had shown that it was due to toxins resulting from trauma. The causes of shock were too vexed a question for discussion that evening. No doubt if the causes were known, it could be better treated or even prevented. However, there were certain physiological features of shock about which all were agreed. Dr. Morgan had mentioned the first three—capillary stagnation, decreased tone of the skeletal muscles and anoxæmia. Capillary stagnation affected blood volume, as was shown by hæmoconcentration, a decrease in venous return (and thus also in cardiac volume) and a decrease in the circulating blood. Capillary stagnation, collapsed veins, depressed respiration and anoxæmia could be observed by the clinician. The laboratory worker found a decrease in the alkali reserve of the blood, but that was partly compensated for by a tendency to acidosis, a reduction in the sodium bicarbonate content of the blood. Actually there was a decrease in the pH leading to partial acidæmia. This gave only partial compensation. But a very important discovery had been made when it was found that there was a rise in the plasma potassium content. Dr. Morgan had referred to the diminution in plasma sodium content; that was apparently allied to the increase in the plasma potassium content, which was perhaps related to the permeability of the cells. The most obvious feature in shock was the much lowered blood pressure. A fall in blood pressure was usually brought about by three factors: myocardial failure, a decrease in peripheral resistance by vasodilatation and a decrease in blood volume. In shock no myocardial failure occurred; the heart beat strongly. There was no vasodilatation; that had been shown. In fact, vasoconstriction was present, although Professor MacDowall, of Glasgow, had stated in one of his articles that vasodilatation was present, and he said that its presence was the main difference between hæmorrhage and shock. Colonel Thomas thought that perhaps in the very late stages of shock, owing to the state of complete anoxia, vasodilatation might be present, and that that might have been what Professor MacDowall meant. Colonel Thomas therefore concluded that the great fall in blood pressure must be due to a fall in blood volume. This might be an actual reduction or an effective reduction. An actual reduction in blood volume might be direct or indirect. Direct reduction was due to severe hæmorrhage, to loss of blood from the vascular system. Indirect reduction was due to transudation of blood plasma into the tissues, to excessive perspiration, to diarrhœa or to vomiting. But as well as an actual diminution in the blood volume, an effective diminution was present owing to dilatation of the capillaries of the skeletal system. It was known that the total blood volume was about five litres. But the capillaries of the skeletal system, if they were all dilated at the one time, could take the whole five litres, and every other system could do the same. Thus, if great dilatation of the capillary vessels in the skeletal system took place, a diminution in the circulating blood occurred. The sequence of events in shock was, first, stagnation of the blood in the capillaries of the skeletal system, followed by an increased volume capacity of the vascular system, leading to an effective reduction in blood volume; that effective reduction led to a reduction in venous return and so to diminished cardiac output; this led to lowered blood pressure and ultimately to anoxia of the tissues, an increase in capillary permeability, transudation and loss of plasma into the tissues. Thus from an effective blood loss events led to actual blood loss. A vicious circle was set up, and once that was so, the process of shock was established and would continue until something was done to break the circle. From the clinical aspect shock presented three phases. They were important, because if one could recognize and treat the first phase, the succeeding phase could often be prevented. The three phases were: the prodromal phase, the intermediate phase and the conclusive phase. In the prodromal phase the pulse was satisfactory and the blood pressure was normal; it was difficult to recognize. The phase was dangerous, because if it was not recognized it would be allowed to pass into the intermediate phase when it was more difficult to treat. The prodromal phase was clinically obvious from the coldness of the patient, his pulse, spasm of the peripheral vessels and tonelessness of the muscles. In this phase there was not the slightest doubt that the arterioles were constricted and that the patient was in good condition. In the intermediate phase the pulse rate was slow and the blood pressure was beginning to fall; in other words, early failure of the vasomotor mechanism was apparent. From that phase the patient would pass into the conclusive phase, in which the blood pressure fell, the pulse rate began to rise and vasomotor failure was established. The condition was not due to the heart or even to the vasomotor disturbance, but to capillary stagnation of the skeletal system.

Colonel Thomas then referred to some practical aspects of shock from the surgical standpoint. Both before and during operation it was essential to use all measures possible to prevent the capillary stagnation and the anoxia which were the fundamental causes of shock. All surgeons knew that shock most frequently supervened in the case of patients who had been subjected to pain or to fear or to too much exertion. (Colonel Thomas did not intend to refer to patients dehydrated by vomiting or diarrhœa.) That was driven home very forcibly overseas when they were receiving patients from the campaign in Greece. All the convoys brought men who were wounded and had been bombed, and who for many days prior to the reception of their wounds had been subjected to a very strenuous time and after being wounded were still harassed. Every man admitted to the hospital showed some degree of shock, sometimes mild, sometimes severe. There was a point of interest in the mild cases; the men were made warm, put to bed and made comfortable with relief of their pain, and in twenty-four hours they were different beings. Colonel

Thomas went on to say that the operating surgeon had certain responsibilities in preventing his patients from becoming shocked. There were three main features which the surgeon had to consider: (i) the duration of the operation, (ii) attempts at hæmostasis, (iii) manipulative dexterity. Colonel Thomas was convinced that the length of the operative procedure had a great deal to do with the development of post-operative shock. He believed that most patients could stand anæsthesia for one hour without showing much distress. But every five minutes over one hour, irrespective of the type of operation being performed, was dangerous to the patient. At the same time, Colonel Thomas did not think that the safety of the patient should be sacrificed to speed. A rough surgeon could do more damage in a short time than a careful surgeon in a longer time. Colonel Thomas was also certain that as complete hæmostasis as possible was absolutely necessary to prevent shock. In cases in which the surgeon expected or suspected shock, regular blood pressure readings should be taken. He should not wait until the patient was back in the ward before intravenous therapy was instituted. Saline solution could be given intravenously in the operating theatre to patients showing any signs of shock. Dr. Morgan had dealt effectively with the treatment of shock; but Colonel Thomas wished to say again, even as a surgeon, that they should not always blame the anæsthetist for the failing condition of the patient; surgeons and anæsthetists both had to take the blame sometimes. In conclusion, Colonel Thomas again thanked Dr. Morgan for his concise and practical discussion of the treatment of shock.

DR. C. N. PATON thanked Dr. Morgan for his masterly handling of the subject. Dr. Paton agreed in general with the points brought forward, but he wished to elaborate on some of the practical points. He had been glad to hear Lieutenant-Colonel Thomas refer to the part the surgeon should play in these problems. Dr. Paton had intended to stress the aspect of shock produced by the operation itself, but this had been done by Dr. Morgan. Dr. Paton went on to say that there was no need for him to dwell on the need for gentleness of manipulation. Even when the patient was not suffering from shock, better attention to such details might lead to better results. The very nature of the operative procedure was often enough to cause shock. Such a procedure was the incorporation of a patient in a plaster spica from the armpits to the ankles; Dr. Paton had often found that two or three hours afterwards the patient was in a severe state of secondary shock. He thought it was probably due to immobilization of the diaphragm; that brought about stagnant anoxia and surgical shock. It was quite impossible for the shock therapist to deal effectively with shock of that type if the operative details were not improved. Dr. Paton further instanced the great traction applied to the lung in several operations for the application of a lung tourniquet; he had found those procedures to be fraught with more shock than any other type of operation. One surgeon had had the idea of dissecting out the hilar structures of the lung and dealing with them one by one; the result had been a great improvement in the condition of such patients. Dr. Paton quite agreed that oxygen and ether were a good anæsthetic combination for patients suffering from shock. He agreed that cyclopropane was the ideal; but its expense and inaccessibility made its use impossible. Dr. Paton used pure gas (nitrous oxide) for six to eight breaths and then introduced ether with oxygen, gradually working up to a concentration of 80%; this he found just as good as cyclopropane. At the chest surgery clinic they had used oxygen and ether as a routine anæsthetic agent for many major operations on the chest, with no bad results. Dr. Morgan had not mentioned one point, the antagonism between cocaine and "Pentothal Sodium". Dr. Paton said that there was a distinct antagonism between them, which made the use of both together impossible. With regard to the question of intubation of patients with intestinal obstruction, Dr. Paton said that it must be done with an inflatable cuff—a circular tube could not block a triangular opening. If the patient began to vomit, it was obvious that his respiratory passages could not be protected. On two occasions Dr. Paton had seen disaster result from intubation without the inflatable cuff. He favoured spinal anæsthesia in most cases of intestinal obstruction; a small amount of "Pentothal Sodium" (five cubic centimetres) enabled him to place the patient in the necessary position for the induction of spinal anæsthesia. The anæsthesia was then continued without difficulty, any further shock being prevented by the great muscular relaxation.

DR. RONALD RAWLE said that he had been impressed with Dr. Morgan's insistence on the early treatment of shock. In effect, in many cases the time to treat shock was before it began at all. That statement was particularly applicable in the case of severe burns, in which treatment by serum and by other means should be instituted immediately. At other times it might not be possible to carry out the ideal treatment of shock suggested, and one was thrown back on second best methods. Dr. Rawle instanced the case of a patient who had been admitted to hospital suffering from hæmorrhagic shock following a severe accident, in which a leg had been run over by a tram. The time necessary to determine the patient's blood group and collect suitable blood for a transfusion would have been too great and therefore other treatment—with serum—was adopted. Dr. Rawle could recall frequent great improvement in the patient's condition after the administration of serum, even in hæmorrhagic shock. Dr. Thomas had insisted on the necessity for beginning treatment in the operating theatre. It had crossed Dr. Rawle's mind that Dr. Morgan had referred to shock coming on when the patient was turned over during operations for abdomino-perineal resection. The administration of serum should be begun before the turning was done, to prevent shock. Dr. Rawle had been pleased to hear Dr. Morgan's rather conservative ideas on the provision of warmth to the patient in a room heated to 75° to 80° F. Dr. Rawle said that they had become rather accustomed to hear recommendations of heroic methods of applying heat. He wondered whether such methods might not do harm. One authority in America (Blalock) had suggested that such procedures might be very bad; arterioconstriction of the superficial vessels might be compensatory, the underlying idea being to keep the blood in the more vital tissues. To cause tremendous vasodilatation to the vessels of the skin might be to defeat one's own ends. Dr. Rawle thought that heat should be applied with discretion in the stage before the blood volume had been restored. Dr. Morgan had referred to the incidence of chest complications, which he said was much higher after local anæsthesia than after general anæsthesia. Dr. Rawle had not found chest complications common after general anæsthesia in Australia. When he was in Manchester the incidence of such complications was high; but the introduction of splanchnic block had brought about a great reduction in it after upper abdominal operations.

DR. A. R. HAMILTON also expressed his thanks to Dr. Morgan. He said that it seemed that the surgeon was to have put upon him the onus of saying when the patient should be treated for shock and when not. Dr. Hamilton always judged from the type of injury when shock was likely to develop, and treated the patient accordingly. He referred to the case of a man who had had both legs from the knees to the ankles crushed by a collision between an omnibus and a tram. Amputation of both legs was needed. The patient was in the operating theatre within an hour, serum was given, and next morning he was sitting up reading the newspaper, and he went straight ahead from then onwards. Dr. Hamilton pointed out that such an injury would produce very severe shock if treatment was not instituted in the early stages. In such cases he saw that the patient was taken to the operating theatre on the bed on which he was put when he was admitted to hospital; the anæsthetic was given and then the patient was transferred to the operating table. Dr. Morgan had mentioned the need for shock teams and shock wards in hospitals; Dr. Hamilton agreed with the suggestion. He suggested that the Federal Council of the British Medical Association in Australia would be doing an excellent thing if it took the matter up with the Hospitals Commission, with the idea of having shock rooms installed in every hospital.

DR. H. C. R. DARLING said that he wished to add his quota of congratulations to Dr. Morgan on his excellent paper. Dr. Morgan had gone through the subject fully and had given one important lesson to the practising surgeon—a little prevention went a long way towards cure. Dr. Darling thought that the reason must be along the following lines: Normally blood of approximately constant composition was circulating in the vascular system. Once the characteristic permeability of the capillary walls developed, plasma passed out of the circulation into the tissues. In shocked patients it therefore followed that if the circulatory system was incapable of retaining its normal contents there was little reason why it should retain allied fluids such as transfusion blood, plasma *et cetera*. Patients still died of shock and no fluid had been devised for the treatment of this malady that was superior to the patients' own blood. Therefore the crucial thing was prevention. In the early stages, how was it possible to diagnose shock? There were the hæmatocrit, plasma protein estimations, the blood viscosity test and the hæmoglobin percentage. The first three tests demanded an expert biochemist; but the hæmoglobin percentage was easily

estimated. No fall in blood pressure would become manifest until the blood volume had been reduced to a considerable degree. Virgil Moon, of Philadelphia, had conclusively demonstrated that blood reached its maximum concentration about four hours before the blood pressure fell to a critical level. Hence the customary estimation of blood pressure must be an unreliable guide wherewith to estimate the degree of shock. The next point to which Dr. Darling referred was capillary dilatation. Was this dilatation universal and uniform? Lewis's work proved that the colour changes in the skin depended largely on the state of the capillaries. Hence the cold pale clammy skin of shock would indicate capillary contraction. In short, during shock, it would appear that the cutaneous and splanchnic capillaries were contracted whilst those of the skeletal muscles were dilated. The change in hue of the skeletal muscles in the shocked patient was well known to the surgeon. One further point to which Dr. Darling wished to refer was in connexion with oxygen therapy. The main carrier for oxygen was the red blood corpuscle, and the efficacy of oxygen would depend on the number of red blood cells present in the blood. Furthermore, the hæmoglobin was virtually saturated and the oxygen content of the cell could not be raised by more than 4%, but the plasma content might be increased from 0·3 cubic centimetre to 2·1 cubic centimetres—a total maximum increase of 15% in the oxygen content of the blood.

DR. C. C. MCKELLAR said that he was grateful for the chance of hearing Dr. Morgan's paper. There were one or two questions he wished to ask. The first was whether there was anything explosive about ether and oxygen. The second had to do with renal lesions following crushing injuries. Dr. McKellar said that the first article on the subject that he had read in the *British Medical Journal* under the heading of "Crushing Injuries and Kidney Lesions" referred to cases in which the limb was not crushed but compressed. Later articles discussed crushing injuries and kidney lesions. Dr. McKellar wondered whether the point was clearly defined—whether such lesions were, in fact, so common in crushing injuries as in simple compression injuries. Dr. McKellar remembered in his student days seeing a patient suddenly rushed back to the ward after having collapsed in the operating theatre. He wondered whether there was anything that could be done in the ward that could not be done in the operating theatre. He also wondered why no operation should last longer than one hour. So long as the patient was being properly treated, he could not understand why every operation should be finished in a certain time. Dr. McKellar remembered watching Dr. George Abbott operating; he took longer than any other surgeon on the staff of the Royal Prince Alfred Hospital. His patients were in the best condition afterwards.

Again, on the same point, Dr. McKellar said that he had read recently an article in which it was stated that an operation lasting two hours on a patient suffering severely from shock was equivalent to a death sentence.

With regard to the application of a plaster spica, referred to by Dr. Paton, Dr. McKellar agreed that a large plaster could be seriously shock-producing and said that Dr. Paton's comments were an argument in support of the necessity for the surgeon to see the patient soon after he had been returned to bed and not wait until the next day.

In conclusion, Dr. McKellar referred to the instruction sometimes given to nurses to hold the patient's jaw forward while he was vomiting; Dr. McKellar thought that was the last thing they should be taught to do, as pulling the jaw forward made an airway and there should be no airway during the actual act of vomiting.

DR. H. J. DALY also thanked Dr. Morgan for his paper. He said he wished to refer to shock occurring during spinal anæsthesia. Such shock was of neurogenic origin. Dr. Daly said this type of shock generally occurred when high spinal or mid-spinal anæsthesia was produced.

He found that the first indication was that the patient ceased talking to the anæsthetist and began to yawn and became pale. This shock occurred about twenty minutes after the spinal anæsthetic was given. The pulse rate might be fast or slow. If a blood pressure machine was used, it would be found that the systolic blood pressure had fallen from perhaps 160 or 170 millimetres of mercury to 100 or less. This fall of blood pressure always accompanied shock in spinal anæsthesia. Soon afterwards vomiting occurred. Formerly ephedrine was used, and it was still in use; but apparently it acted on the preganglionic fibres, and in spinal anæsthesia some of these were often paralysed. "Neo-synephrin", on the other hand, acted peripherally. Dr. Daly thought that "Neo-synephrin" was the most valuable drug used at the present time to combat such shock. Dr. S. V. Marshall had used it in Sydney about five years earlier, and had pointed out its value. Its advantage over ephedrine was that it would bring the patient back to good condition after he had passed into the stage of shock. The dose was 0·5 cubic centimetre given intramuscularly. If the patient's blood pressure was falling, another dose of "Neo-synephrin" should be given intravenously. Dr. Daly uttered a warning against using more than three or four minims; he said that if larger amounts were given, the patient's blood pressure would rise very high. Some years previously he had had a patient whose systolic blood pressure had fallen to 90 millimetres of mercury; he gave him 0·5 cubic centimetre of "Neo-synephrin", and five minutes later the blood pressure had risen to 120 millimetres of mercury; ten minutes later it had risen to 160, and some minutes later it rose to 220. After that Dr. Daly did not take further blood pressure readings—he was too frightened; but the patient seemed none the worse.

DR. GEORGE BELL said that Dr. Morgan had wisely stressed the importance of preventing shock, and in that regard he (Dr. Bell) referred to the value of efficient first aid, which could play a great part in the prevention of shock. All those present knew what had happened when the Thomas splint was introduced in the first aid treatment of fractures of the femur during the last war in France. Patients arrived at the casualty clearing station in much better condition than previously. Dr. Bell had been told that one of the great dangers in first aid lay in the temptation to do too much; first aid personnel were apt to roll and move the patient about. This was wrong; it increased shock. Dr. Bell went on to refer to Dr. Morgan's remarks about team work. He said that he could remember one very efficient team; it consisted of Taylor and Geoffrey Marshall, of Guy's Hospital. Marshall had been a physiologist before the last war, and had done a considerable amount of work on fall of blood pressure in high amputations of the thigh; he had found that gas and oxygen anæsthesia gave better results than ether. Dr. Bell remembered him in a team doing abdominal surgery; Marshall gave the anæsthetic gas and oxygen. Taylor used some local infiltration anæsthesia, and Marshall, while the surgeon was finishing, gave a transfusion of whole blood. Dr. Bell pointed out that it should be possible for an anæsthetist to do both things at once. With regard to lung blast, Dr. Bell said that a few days previously he had been discussing the subject with Colonel Hailes. Colonel Hailes had warned him that lung blast would occur if cities were bombed, and that anæsthesia was contraindicated. Intravenous administration of fluid was dangerous to patients with lung blast, as the patients easily became water-logged. With regard to chest surgery, Dr. Bell said that apparently thoracic injuries had not been very numerous in the Middle East. Colonel Hailes had stressed the great importance of the prevention of shock, particularly in the presence of an open pneumothorax sucking wound, by the insertion of sutures and closure of the wound at the first aid post. Such patients should be closely watched during the first twenty-four to forty-eight hours, and if they showed dyspnœa the chest should be aspirated. In chest surgery, close cooperation was necessary, not only between the surgeon and the anæsthetist, but also between surgeon, anæsthetist and physician as well. It had been Dr. Bell's experience that physicians were very helpful in the treatment of acute thoracic lesions. In conclusion, Dr. Bell said that Colonel Hailes had stressed the importance of hæmoglobin estimations, especially in severe burns.

Dr. Morgan, in reply, thanked the various speakers for the gaps they had filled in and for the suggestions they had made. He said that in a paper such as that he had presented, gaps were inevitable; the subject was huge. One of his difficulties had been to decide what could be left out. Dr. Morgan was grateful to Lieutenant-Colonel Thomas for his account of practical experiences and for giving those present the benefit of the knowledge he had gained. Colonel Thomas had mentioned three points: duration of the operation, the ensuring of adequate hæmostasis and careful handling of the tissues. Dr. Morgan said that all anæsthetists who took blood pressure readings during operations would like to add a fourth point: the covering of raw surfaces. During the removal of a breast, for example, the covering of raw surfaces with warm saline packs was sufficient to reverse a fall in blood pressure and a rise in pulse rate. Dr. Morgan said that he was grateful to Dr. Paton for his remarks. He did not know whether he had misunderstood something Dr. Paton had said concerning an antagonism between the barbiturates and cocaine, so that they could not be used together. Dr. Morgan said that he thought it was

that very antagonism which made it possible to use them together. The exhibition of "Pentothal" was the recognized treatment for cocaine poisoning. The use of the barbiturates in conjunction with large quantities of local anæsthetic agents destroyed the toxicity of such anæsthetic agents and made local anæsthesia possible. Dr. Morgan went on to say that the point raised by Dr. Rawle was important; he referred to the association of shock and hæmorrhage. In such a case it was known that blood was the requirement. But the use of serum, or in the absence of serum the use of saline solution, while the patient's blood was being typed and suitable blood was being collected, was frequently a life-saving measure. Dr. Morgan remembered one patient who had been brought to hospital by ambulance in such a condition that the ambulance men seemed inclined to take him straight to the morgue. While the patient's blood was being typed glucose solution was run in, and by the time the transfusion was ready the patient's condition had considerably improved. The indications were that local anæsthetic agents should not be used to prevent complications when they were likely to occur. Dr. Morgan thanked Dr. Darling for his remarks. With regard to Dr. McKellar's inquiry as to whether ether and oxygen formed an explosive mixture, Dr. Morgan said that in mixtures varying from 3·8% to 88% the combination was most explosive. It was generally used in the absence of any exposed flame. Dr. McKellar had also asked about kidney lesions in crush and compression injuries. Dr. Morgan said that if the theory was accepted that kidney lesions were primarily due to oxygen want in the kidney cells, then such lesions would be present in all types of shock. Dr. McKellar's third point had had to do with a patient whom he had seen being rushed back to the ward from the operating theatre. Dr. Morgan said that he thought that the only idea in people's minds when such a procedure was carried out was the avoidance of a possible visit to the coroner's court; he knew no other reason. All treatment that could be carried out in the ward could be better carried out in the operating theatre. To hurry a patient away in such a manner was quite contrary to the anæsthetist's wishes. Dr. Daly had referred to shock in spinal anæsthesia. Dr. Morgan said that he had not mentioned it because that was primary shock, not secondary shock. Dr. Daly's remarks had been to the point; "Neo-synephrin" had made the anæsthetist much more comfortable when he was using spinal anæsthetics. Dr. Daly's references to the prevention of shock had been good. Dr. Morgan would say that in all cases in which experience had taught them to expect shock, they should institute shock therapy, until such time as properly equipped shock teams had been developed, capable of making the various estimations that would provide information for the early diagnosis of shock in the prodromal stage mentioned by Dr. Thomas. Finally, Dr. Morgan stressed the need for properly equipped shock wards—properly equipped both from the point of view of personnel and from that of treatment. It was time that such wards were in existence in Australia. They did not necessarily entail extra expense, and he was certain that great benefit would be derived from them, both in civilian and in wartime emergencies. Dr. Morgan also stressed the necessity for the institution of shock treatment in all cases in which previous experience had shown that shock was likely to develop; he referred particularly to burns, hæmorrhage and leg injuries. Dr. Morgan also suggested that wherever it was possible, the quantitative tests that had been mentioned should be used and asked for more, so that the pathological departments would become used to carrying them out. The hæmatocrit reading was simply made, and hæmoglobin estimations also gave much information. Much difficulty was associated with the carrying out of plasma protein estimations in a time sufficiently short for them to be of value in the diagnosis of shock, particularly when water loss and protein loss were great. But overseas there was available a compact apparatus for carrying out this estimation, and if the import authorities would permit it the apparatus would be in Australia shortly. The present system was rather like setting out to carry out a blood count without a proper counting chamber. In conclusion, Dr. Morgan once again thanked those present for the interest taken in his paper and the speakers for their valuable contributions.

DR. W. F. SIMMONS, from the chair, said that those present had appreciated the effort and time spent by Dr. Morgan in the preparation of his valuable contribution, as had been proved by the various comments of those who had spoken. Dr. Simmons said that he thought that Dr. Morgan could take hope that one thing at least that would follow the present national emergency was that most hospitals would be equipped once and for all to treat shock in its various forms. That matter had been impressed upon them. For that reason some good might come that would be of lasting value to shock therapy in the future years of medical practice in Australia and in other countries that had suffered from the ravages of war.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 21, 1942, at the Royal Prince Alfred Hospital, Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital. Part of this report was published in the issue of August 22, 1942.

Hæmangioendothelioma of the Nasal Septum.

DR. J. J. WOODBURN showed a male patient, aged nineteen years, who had been admitted to hospital on October 31, 1941, complaining of blocking of the left nostril of three weeks' duration, and of bleeding from the nose of six weeks' duration. The blocking had occurred suddenly, and from a biopsy a diagnosis of hæmangioendothelioma was made. Examination revealed the left nostril to be completely blocked by a large polypoid growth.

Operation was undertaken on the day of his admission to hospital, "Pentothal Sodium" being the anæsthetic agent. The growth was removed from the septum. On November 4, radium was applied to the left nostril, to remain in position for 144 hours; one "unit three" needle was used, consisting of three milligrammes of radium in a rubber tube with 0·5 millimetre of platinum filtration. The needle was removed on November 10. Dr. Woodburn said that the patient was cured. At the time of the meeting he was in good health and had gained in weight; his nose was quite free.

Macroscopic examination of the tumour showed it to be an oval piece of tissue three centimetres in length. The surface was very vascular. On section the tissue was uniformly white in appearance, but in one part a considerable amount of hæmorrhage had taken place into its substance. On microscopic examination, the tumour was seen to be an hæmangioendothelioma, which appeared to be more active than normal; in one area hæmorrhage and thrombosis of the vessels were apparent.

Carcinoma Treated with Radon.

DR. P. D. BRADDON showed two patients to refute certain teachings to medical students which had come to his knowledge recently. The first patient was a woman, aged seventy-eight years, who had presented herself twelve months earlier with an early carcinoma of the right side of the tongue. It measured two centimetres in diameter, and was treated by a permanent gold seed radon implant to a dose of 7,000 *r*. She had never been admitted to hospital, as her implant had been inserted under local anæsthesia by the application of a 10% solution of cocaine, and she had gone home immediately afterwards. She now presented a tongue in which it was difficult to discern where the lesion had been, no secondary deposits in glands had developed, and it was not intended to ablate her glands at her age and in view of the fact that the growth was early. Dr. Braddon's experience abroad in 1939 was that large numbers of early carcinomata of the tongue in elderly patients well on in the eighth decade or later could be treated as a local disease, and that nothing further was necessary. Occasionally a patient would be lost; but Dr. Braddon showed this particular patient as one of a series of twelve treated in the last two and a half years, all of whom had almost perfect tongues, had never been in hospital, had undergone no surgical operation and were in good health for their age. Two of the patients had been treated well over two years earlier. Dr. Braddon said that he had ascertained that students had been told that radon seeds were "a menace, and that there should be a law to prevent their use". He could only conclude, if this was true, that the lecturer was referring to glass seeds, of ill fame and no filtration. Modern gold seeds, though exceedingly small, had the same filtration as the average radium needle. Radon seeds provided an excellent, indispensable and accurate technique, and could often be used where radium was contraindicated or impossible to apply.

Dr. Braddon's second patient had presented himself nine months earlier with a large, ulcerated carcinoma of the dorsum of the right hand, four centimetres in diameter. At the time of the meeting he had an excellent hand, and close inspection was needed to detect which hand had borne the original lesion. The skin was pliable, soft and free, and exhibited practically no scarring. The tendons were free

and the function of the hand was 100%. The treatment consisted of the application of a radon mould at one centimetre distance to a dose of 6,000 *r* over fourteen days; the biological or time factor was vital to success, as the skin of the dorsum of the hand of old patients was vulnerable, thin, shiny and atrophic, and had a poor blood supply. The lesion received the usual dose of 6,000 *r* to an epithelioma of the skin, but the dose was spread over double the usual time. At the Royal Prince Alfred Hospital Radium Clinic large numbers of such patients were treated annually, with almost 100% success. Radio-necrosis never occurred. The patients had the mould fitted to exact measurements and distance, and radon was applied to give a dose of 3,000 *r* over seven days. At the end of this period they returned to the clinic and a fresh application of radon was made to the mould to give another 3,000 *r*; thus a total of 6,000 *r* was given continuously over fourteen days. Hands with large growths were always splinted during treatment. Dr. Braddon said that the patient was shown to refute recent teaching that radium (or radon) should never be used on growths of the dorsum of the hand. Such a statement was utterly erroneous. Not only was radium or radon not contra-indicated, but, properly used, it gave far the best result with advanced growths of any method of treatment, and saved many a useful hand.

Osteogenesis Imperfecta.

DR. L. J. WOODLAND showed two patients suffering from *osteogenesis imperfecta* (*fragilitas ossium*, osteopsathyrosis). The first patient, a female, aged thirty-two years, had been referred to the orthopædic department of the Royal Prince Alfred Hospital with the complaint of backache and pain in feet of several months' duration. Her past history was that she had been prone to minor joint injury, but had never sustained a fracture. She had begun to go deaf at the age of twenty years and now relied entirely on lip reading. She was the youngest but one of thirteen children, all the rest of whom were normal. Her father had blue sclerotics, and his head was the same shape as that of the patient. He was not deaf and was not subject to fractures. The patient had had three children, two of whom were normal; the third was the second patient shown at the meeting.

On examination, the patient was a small, thin, intelligent woman; her weight was six stone four pounds and her height four feet ten inches. Blue sclerotics, hypotonia of muscles and the typical shape of the head were noted. Lumber scoliosis and early foot collapse were found to be the cause of her backache and the pains in her feet. The predisposing cause of these deformities was muscle hypotonia. The treatment was support and rest with suitable graduated exercises.

Dr. Woodland said that this patient illustrated all the features of the non-brittle type of this disease. (i) Blue sclerotics became greyer as age advanced. This so-called "Wedgwood blue" was more noticeable near the iris. Blueness was a more dominant character than brittleness. Of people with blue sclerotics, 50% had bone fragility. Brittleness did not occur without blueness. (ii) Impairment of growth was due, not to fractures, but to early synostosis of the epiphyses. (iii) The characteristic shape of the head was due to bulgings at the occipital and parietal eminences. The latter caused the ears to face downwards and outwards. The face appeared to be small and triangular, owing to the increased transverse diameter of the calvarium. (iv) Increased liability to joint injury was more pronounced in the non-brittle type of bone fragility. This liability to sprains was said to be due to the laxity of ligaments, but really muscle hypotonia was just as important a factor. (v) Deafness was rare before the age of twenty years. This progressive otosclerosis occurred in 33% of persons with blue sclerotics. It attacked equally those with brittle and those with non-brittle bones. (vi) The disease was an hereditary disease, which affected both sexes equally; it was a dominant Mendelian character conveyed by either sex.

Dr. Woodland's second patient was a female, aged five years. She had first been examined at the Royal Alexandra Hospital for Children sixteen months earlier. Her parents complained that she had a peculiar gait, of two months' duration. She complained of intermittent pain in her limbs, feet and back. Her dentition had been normal. She had commenced to walk at the age of fourteen months, and had "always been weak on her ankles". The peculiar gait was due to a fracture of the shaft of the first left metatarsal bone. Since then she had had eight fractures, affecting the metatarsals of both feet, the phalanges of the fingers, the tibia and fibula, the medial epicondyle of the humerus, and the acromion process. At the time of the meeting she had a fractured occipital bone.

Dr. Woodland said that the child showed the typical signs of the adolescent type of the disease, more correctly termed *osteogenesis imperfecta tarda*. She had blue sclerotics, and the shape of her head and face was characteristic. Hypotonia of the muscles and laxity of the ligaments permitted ligamentous strain and joint instability. This caused the symptoms of pain in the limbs and back and the signs of *genu recurvatum*, knock knees, flat feet and general bad posture. Fragility of the bones was a feature. The fractures were mainly subperiosteal and so did not cause much pain; on this account such fractures might be overlooked, and so lead to deformity. The fractures united readily; the callus was as dense as in fractures in normal people. Fractures seldom appeared before the age of three years in this type of the condition. The blood calcium content was 12 milligrammes per 100 cubic centimetres, and the blood phosphorus content was 4·1 milligrammes per 100 cubic centimetres. In this condition the blood calcium and phosphorus contents might be higher or lower than normal.

The fractures were treated in the usual manner. With regard to prognosis, Dr. Woodland said that fractures occurred infrequently after the age of sixteen years. As the shafts of bones tended to sclerose at about that age, so in 33% of cases otosclerosis began to develop.

Dr. Woodland then discussed the radiological features of the lesion. He said that the long bones had slender shafts with expanded metaphyses and epiphyses. The shafts were dense relative to the extremities, in which osteoporosis was present. Many cross striations were seen. In the skull were seen irregular ossification and islands of denser bone in a poorly calcified matrix, the so-called Wormian bones. Areas of thin bone were seen at the vertex and at the parietal and occipital eminences; bulges were apparent at these sites.

Dr. Woodland said that in the normal development of bone there were three factors: (i) a proper cartilaginous or membranous scaffold, (ii) the deposition of necessary inorganic salts, (iii) the presence of a sufficient number of normal bone-forming cells. In the disease under discussion the essential feature was the failure of differentiation of osteoblasts. There were four clinical types of the disease. (i) The fœtal type: the child was usually stillborn. It probably died at birth from cerebral injury. Hundreds of fractures might be present, some already united. (ii) The infantile type: the child usually survived for a year or two; the bones fractured on being touched. (iii) The adolescent type, illustrated by the second patient shown by Dr. Woodland. (iv) The non-brittle type, illustrated by the first patient.

Madelung's Deformity.

Dr. Woodland also showed a female patient, aged nine years, who had Madelung's deformity of the left wrist. She was born with a nævus over the distal end of the left radius on its posterior aspect. At the age of three months she had been treated with a half dose of irradiation from the posterior aspect and a half dose from the anterior aspect. This treatment cured the nævus, which was apparently fairly large. The deformity was first noticed at the age of five years. The child had first come under observation at the orthopædic department two years earlier. Plaster casts made at intervals since then revealed very little change. X-ray examinations showed that the condition was becoming worse.

On examination, the characteristic signs of Madelung's deformity were seen. They were the following: (i) Shortening of the radius, which caused radial deviation of the wrist. (ii) Prominence of the head of the ulna. The apparent dorsal displacement of the ulna was due to anterior subluxation of the carpus. (iii) Limitation of dorsiflexion, which was due to increased anterior inclination of the concave articular surface of the radius. X-ray examinations showed that the deficiency in growth of the left radius was due to a lesion at the radial epiphyseal line. The metaphysis was sclerotic, irregular and to some extent cupped. The right radius was normal.

Dr. Woodland commented that the deformity was probably due to the X-ray therapy, which had interfered with the radial epiphyseal line. The epiphysis usually appeared at the age of about two years and united at the age of twenty-four years. It was necessary to exclude syphilis, trauma, epiphysitis and congenital lack of development of the epiphysis. The prognosis was that the deformity would become worse until fusion of the ulnar epiphysis occurred; this epiphysis appeared at the age of eight years and united at about twenty-four years. The prognosis with regard to function was generally favourable. The treatment was the use of a right splint to check the deformity. In early adult life subperiosteal resection of the lower end of the ulna gave good results.

Congenital Abnormalities of the Foot.

Dr. Woodland then showed a female patient, aged three months, with congenital *talipes calcaneo-valgus* and *metatarsus valgus* of the right foot. The abduction of the forefoot caused a pronounced groove at the cubo-metatarsal joint. An X-ray examination revealed normal ossification for the infant's age. She was treated by manipulations and the use of adhesive plaster splints. "C" and "E" heels would be necessary when the infant commenced to stand.

Dr. Woodland remarked that a moderate degree of *talipes calcaneo-valgus* was relatively common. It was practically self-correcting, because the calf muscles were much stronger than the dorsiflexors. The extreme type of *talipes calcaneo-valgus* was rare. It was usually associated with subluxation at the talo-calcaneal joint and was really a congenital *pes plano-valgus*. In some cases the treatment of this type of lesion was difficult. In the patient shown the *talipes calcaneo-valgus* was pronounced, but not extreme. The dominating feature was the abduction of the forefoot—*metatarsus valgus*. In his experience it was a rare condition.

A Corrective Shoe in Foot Collapse.

Dr. Woodland then showed a corrective shoe and demonstrated its use in a case of foot collapse. The features of the corrective shoe were the addition to a correct shoe of a Thomas heel and of a comma-shaped metatarsal bar, as used by Hauser. The Thomas heel corrected the valgus tilt of the calcaneus and provided support to the talo-navicular joint. The Hauser metatarsal bar was one-eighth of an inch higher on the lateral side than on the medial side, and it ended laterally five-eighths of an inch from the lateral margin of the sole. This bar corrected the supination of the forefoot, reestablished the transverse arch and so corrected the splaying of the forefoot.

Dr. Woodland said that the use of a corrective shoe was only one part of the treatment of foot collapse. Manipulation, exercises and physical therapy were all used. Education in correct walking was absolutely essential. The results were excellent.

Correspondence.

MEDICAL CERTIFICATES.

SIR: We, the administrative officers of the Wirraway Benefit Society, feel that certain facts should be brought to the notice of the members of your profession, because they have a threefold interest, yours, ours and a national one.

Employees working under the aircraft award are granted annual holiday leave on full pay, may also claim two working weeks' sick leave per annum on full pay and may also draw up to twelve weeks *per annum* from our society at 25s. a week in addition (if incapable of attending work through sickness or accident), the subscription for the latter being but sixpence per week.

It is unnecessary to labour the point, that a man who secures a medical certificate is financially better off during that period than when working. You notice we do not say "a man who is too ill to work", but "a man who secures a medical certificate", which, in so many cases, is not an incapable man, but merely an unwilling to work man.

Under the Security Regulations, any aircraft employee absent from his employment without a medical certificate, may find himself marked "A.W.L." and thereby at the disposal of the military authorities—hence the great demand for medical certificates.

We have never questioned any medical practitioner's diagnosis, but occasions have arisen, through information from sources inside the works, when it was necessary to contact the doctor concerning the statement printed boldly on all our medical certificate forms—namely, He was incapable of attending work.

In 98% of such contacts, the doctor has said either that he never noticed the sentence or has given some similar explanation, but they have, one and all, stated the patient was capable of working, but was advised to have a holiday.

The writers of this letter have always worked harmoniously with the profession and the British Medical Association; we have received nothing but the most courteous treatment from them, and because of this, we feel emboldened to ask the medical profession in general, and your readers in particular, to strike out that "incapable of attending work" clause unless it is fully warranted.

So many doctors have told us that our members visit them describing symptoms which point to nervous debility, neurasthenia or a similar condition, the prevalence of which is greater during war than in normal times. These same doctors have told us that although they cannot find much the matter with these people, they recommend them to get away into the country for a fortnight; perfect advice if we may presume to say so, but we earnestly entreat you to refrain from signing (for any compassionate reason) that such people are incapable of attending work; if such a patient should have a holiday—well and good—but not on the grounds of being incapable, because under our constitution we may only pay sick claims when, and only when, that condition exists.

By doing this, you will soon reduce the demands on your valuable time, you will be doing us a great service, and last, but not least, you will make the way of the "absentee" a harder road to tread.

Yours, etc.,

Countersigned:

D. HUTCHINGS,
Honorary Treasurer.

E. BALL,
Honorary Secretary, Wirraway Benefit Society.

C.o. Box 779H,
Elizabeth Street P.O.,
Melbourne.

Medical Practice.

A RADIO TALK FROM LONDON.

THE Australian Broadcasting Commission announces the receipt of a cable from the British Broadcasting Corporation to the effect that the Secretary of the British Medical Asscciation will broadcast from London on the subject of the post-war medical plans of a committee of the Association. The broadcast will take place in the "Calling Australia" session on Saturday, September 12, at 5.45 o'clock p.m. eastern (Australian) standard time on the alternative national network, that is, from stations 4QR in Queensland, 2BL, 2NC and 2CR in New South Wales, 3LO and 3WV in Victoria, 5CL in South Australia, 6WN in Western Australia and 7ZL in Tasmania.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 225, of August 20, 1942.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

Temporary Squadron Leader J. C. Fulton (1219) is granted the acting rank of Wing Commander whilst employed as a Wing Commander with effect from 16th March, 1942.

The following Flight Lieutenants are granted the acting rank of Squadron Leader whilst employed as Squadron Leaders with effect from the dates indicated: R. S. Robinson (1422), 1st April, 1942; H. T. Harvie (1924), 26th March, 1942; E. V. W. Pockley (3752), 22nd January, 1942.

Flying Officer J. C. Watson (1868) is granted the rank of Flight Lieutenant whilst employed as a Flight Lieutenant, with effect from 1st June, 1942.

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Gordon Sylvester Alexander Shearwin (6332), Joseph Hart Wall (6333), 29th June, 1942; Egbert Dennis, M.B., B.S. (6288), 8th July, 1942; Raymond Eddington Woods, M.B., B.S. (6330), 10th July, 1942.

No. 57280 Aircraftman Class I Frank Heuston Lavery (6124), who attended No. 11 Equipment Course, is appointed to a commission on probation with the rank of Pilot Officer with effect from 11th July, 1942.—(Ex. Min. No. 119—Approved 18th August, 1942.)

Reserve: Medical Branch.

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from dates indicated: Alexander Clement Herrington, M.B., B.S. (6327), 10th July, 1942; Donald Stuart Kidd, M.B., B.S. (6325), 12th July, 1942.—(Ex. Min. No. 117—Approved 18th August, 1942.)

CASUALTIES.

ACCORDING to the casualty list received on August 25, 1942, Captain R. I. Mannion, A.A.M.C., Taringa, Queensland, Captain K. C. M. Madden, A.A.M.C., Woombye, Queensland, and Captain D. G. Picone, A.A.M.C., Cooroy, Queensland, are reported to be missing overseas.

Obituary.

LESLIE GEORGE GRIFFITHS.

WE regret to announce the death of Dr. Leslie George Griffiths, which occurred on August 3, 1942, at Isfahan, Iran.

MAURICE PATRICK MACGILLICUDDY.

WE regret to announce the death of Dr. Maurice Patrick MacGillicuddy, which occurred in August, 1942, at London, England.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Pullen, Wallace James, M.B., B.S., 1939 (Univ. Sydney), N27148 Captain W. J. Pullen, Regimental Medical Officer, 36 Battalion, Australian Military Forces, Australia.

Chesher, Rupert George, M.B., 1941 (Univ. Sydney), District Hospital, Parramatta.

Allen, Loraine G., M.B., B.S., 1942 (Univ. Sydney), District Hospital, Bathurst.

Edye, John Andrew, M.B., B.S., 1942 (Univ. Sydney), 13, Kirkoswald Avenue, Mosman.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered pursuant to the provisions of the *Medical Act*, 1918, of Tasmania as duly qualified medical practitioners:

Booth, Robert Philip, M.B., B.S., 1932 (Univ. Melbourne), Dip. Anæs. R.C.P. & S., 1937, Rosetta.

Baldwin, Richard John, M.B., B.S., 1942 (Univ. Melbourne), Launceston Public Hospital, Launceston.

Morris, Heather Byers, M.B., B.S., 1942 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Bretherton, Kenneth Wilbur, M.B., B.S., 1942 (Univ. Melbourne), Launceston Public Hospital, Launceston.

Grove, Graeme Lindsay, M.B., B.S., 1940 (Univ. Melbourne), Brisbane Street, Launceston.

Zerman, Percy, M.B., B.S., 1942 (Univ. Melbourne), Launceston Public Hospital, Launceston.

Rae, Douglas John, M.B., B.S., 1942 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Purton, Herbert Culvernon, M.B., B.S., 1942 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Wilson, Jean Wilson (formerly Hill), M.B., B.S., 1941 (Univ. Melbourne), Launceston.

Fitzpatrick, Leigh Alan, M.B., B.S., 1942 (Univ. Melbourne), Launceston Public Hospital, Launceston.

Medical Appointments.

Dr. Reginald Frank May and Dr. Charles Hugh Hembrow have been appointed Members of the Masseurs Registration Board of Victoria until June 30, 1945, pursuant to the provisions of section 4 of the *Masseurs Registration Act*, 1928.

Dr. Brian Windeyer, a graduate of the University of Sydney, has been appointed professor of radiology in the University of London.

Books Received.

"The Nursing of Nervous Patients", by John Bostock, M.B., B.S. (London), M.R.C.S. (England), L.R.C.P. (London), D.P.M., F.R.A.C.P.; 1942. Brisbane: University of Queensland. Crown 8vo, pp 176.

"Spontaneous and Experimental Leukæmia in Animals", by Julius Engelbreth-Holm, M.D., translated from Danish into English by C. L. Heel; 1942. Edinburgh: Oliver and Boyd. Medium 8vo, pp. 260 with 44 illustrations. Price: 15s. net.

Diary for the Month.

SEPT. 8.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 11.—Queensland Branch, B.M.A.: Council.
SEPT. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 16.—Western Australian Branch, B.M.A.: Branch.
SEPT. 17.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 24.—New South Wales Branch, B.M.A.: Branch.
SEPT. 24.—South Australian Branch, B.M.A.: Branch.
SEPT. 25.—Federal Council of the B.M.A. in Australia: Meeting in Melbourne.
SEPT. 25.—Queensland Branch, B.M.A.: Council.
SEPT. 25.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility unless such a notification is received within one month.

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